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Original Communications

THE SURGICAL TREATMENT OF AORTIC STENOSIS

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DEFINITION

THE term aortic stenosis refers to a significant narrowing of the orifice of the aortic valve. While it may be related to the presence of a constricted valve annulus of congenital or acquired origin, it usually is the result of intrinsic stiffening of the leaflets or cross fusion of their free margins (commissural obliteration).

HISTORICAL OBSERVATIONS

Riverius,[1] in 1647, apparently was the first to describe the pathologic changes of what undoubtedly was calcareous aortic stenosis, and to correlate them with the clinical features of a weak pulse and diminished arterial pulsations. Rayger,[2] Cowper,[3] Vieussens,[4] and Morgagni[5] have contributed early to the understanding of this entity.

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ETIOLOGY

It is believed that clinical aortic stenosis may be caused by congenital deformation, by arteriosclerotic changes in the aortic wall and valve leaflets, by the

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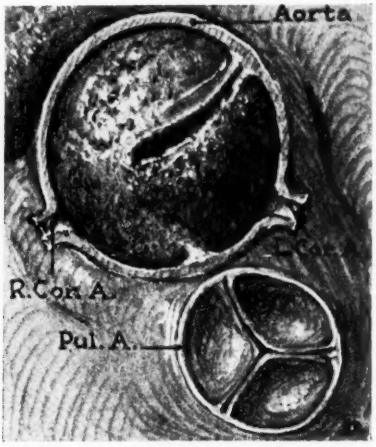

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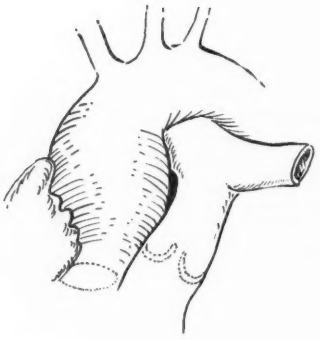

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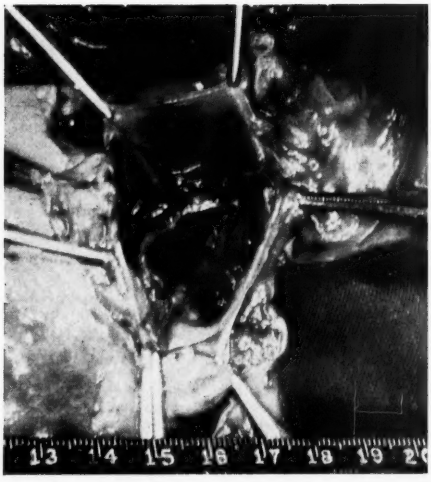


D.

Fig. 1.—*A*, Caplike congenital fusion of aortic valve cusps. (From Bailey, Surgery of the Heart, courtesy Lea & Febiger.)

B, Asymmetric congenital fusion of aortic valve cusps with only one commissure open. (From Taussig, Congenital Malformations of the Heart, courtesy Commonwealth Fund.)

C, Poststenotic dilatation of ascending aorta in congenital aortic stenosis. This also may be observed in certain patients with long standing acquired stenosis. (From Bailey, Surgery of the Heart, courtesy Lea & Febiger.)

D, Congenital fusion of two aortic leaflets producing a bicuspid valve. Note rudimentary raphe dividing the larger cusp. This patient exhibited aortic regurgitation as well as stenosis.

ulcerative and healing phases of bacterial endocarditis superimposed upon a previously abnormal valve, and, most commonly, by the effects of rheumatic valvulitis, alone.

Congenital Aortic Stenosis.—It is clearly evident that aortic stenosis may be of congenital origin. In these cases, there may be a generalized hypoplasia of the first portion of the aorta and the valve, often associated with a subvalvular stenosis. In other instances, the valve may present a "cap-like" or "megaphone-like" fusion of the cusps, forming a funnel-shaped diaphragm with a small centrally or eccentrically located orifice. The latter may represent an incomplete obliteration of one commissure (or two). In congenital cases, a post-stenotic dilatation of the ascending aorta is usual (Fig. 1, *A, B, C*). This phenomenon also may be observed sometimes in cases of long-standing advanced acquired aortic stenosis.

Another type of congenital aortic stenosis is that which is associated with a bicuspid aortic valve. However, not all bicuspid aortic valves are stenotic; some are associated with regurgitation. In most of these valves, two of the commissures will be normal but one (usually the anterior) will be found to be underdeveloped or incomplete. It may be represented by a rudimentary raphe dividing the larger of the cusps (Fig. 1, *D*). This type of valve frequently is found in association with coaretation of the aorta. Indeed, Reifenstein and his associates[6] have reported that 42.7 per cent of their series of 104 patients with coaretation also had a bicuspid aortic valve.

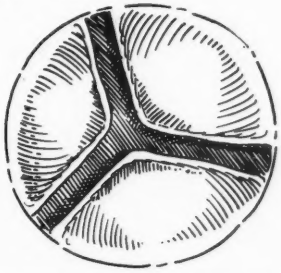


Fig. 2.—Arteriosclerotic aortic stenosis does not produce obliteration of the commissures. Instead, the leaflets may become stiffened and springy but preserve their natural anatomical contours. (From Bailey, Surgery of the Heart, courtesy Lea & Febiger.)

Arteriosclerotic Aortic Stenosis.—Whereas formerly it was felt generally that acquired aortic stenosis, especially when associated with calcification, was usually of arteriosclerotic origin,[7, 8] today, most authors feel that the majority are caused by rheumatic valvulitis.[9-12] In our experience, approximately one-half of the patients with acquired aortic stenosis also present coexisting indisputable rheumatic disease of the mitral valve. Presumably, both valves in this group of patients have been affected by the same etiological process. Furthermore, since the majority of the remaining patients present a spectrum of aortic pathology which is identical with that observed in combined aortic and mitral valvular disease, we are inclined usually to indict the rheumatic state for the initiation of acquired aortic stenosis. The frequency of the occurrence of calcification in the involved valve is not pertinent since calcification develops

occasionally in congenitally deformed, and, very often, in rheumatic stenotic valve lesions. It is found to a greater or lesser extent in over 90 per cent of stenotic aortic valves examined digitally during transaortic surgery or at autopsy.

In general, we have felt that the arteriosclerotic process does not produce commissural fusion but merely stiffens the nondeformed valves in a position of incomplete closure[13] (Fig. 2). Thus, some insufficiency must coexist in practically every one of these valves.

Aortic Stenosis Resulting From Subacute Bacterial Infection.—While such lesions undeniably exist due to the organization of the vegetations and the fibrosis which ultimately may develop upon the healing ulcerative lesions, they are relatively rare. Since bacterial infection apparently always must be superimposed upon a valve already deformed by congenital malformation or rheumatic distortion, the structural abnormality tends to follow the pattern of the primary pathology and is amenable to therapy appropriate for it.

Rheumatic Aortic Stenosis.—The pathogenesis and pathology of rheumatic aortic stenosis are in most respects analogous to that of rheumatic mitral stenosis, so well described by Rokitansky[14] and Magarey.[15] It appears to occur clinically about one-fifth as frequently as the latter disease, and shows a preponderant predilection for males. Aortic stenosis was diagnosed in 365 (32.5 per cent) of 1,585 patients operated upon for rheumatic valvular disease at the Hahnemann Hospital in Philadelphia. Rheumatic vegetations developing upon the free margins and the lunulae (zones of leaflet contact) of the cusps produce surface irregularities upon the ventricular aspect of the valve. Blood flowing through the valve passageway develops a peripheral turbulence due to this roughening of the walls of the channel. In spite of the rapid blood flow, a certain amount of surface deposition of platelets and fibrin results (perhaps related in part to defibrination of the flowing blood). Organization, both of the vegetations (really minute thrombi) and of the fibrinous deposit upon the valve, applies a thin layer of scar tissue upon the ventricular aspects of the cusps and over their free margins. Until the surface irregularities have become buried completely within additional subsequently laid down lamellae of scar tissue and incorporated within the thereby thickened valve substance, the process must recur from time to time even in the absence of any further rheumatic exacerbation. Obviously, an exacerbation of the rheumatic state may reproduce a new cycle of surface irregularity, turbulent flow, fibrinous deposition, organization, etc. Thus, "new" tissue of fibrous character becomes added to the ventricular aspect of the leaflets. To a lesser extent, the process may "spill over" onto the concave aspect of the valve and bring about thickening of the aortic surfaces of the cusps.

At the same time, the fibrinous exudate, due to the incessant movement of the cusps, tends to become heaped up and to bridge the most peripheral extremities or angles formed by the adjacent cusps at their aortic attachments. As this fibrin becomes organized, the peripheral portions of the free margins of the cusps become adherent. Ultimately, a centripetally progressive adhesive valvulitis results in extensive cross fusion of the free edges and the lunulae of the leaflets (Fig. 3, *A, B, C, D, E*).

Logically, this process, if symmetrical, gradually should reduce the valve orifice to a tiny central equilateral triangular opening. However, progressive centripetal cross fusion of the leaflets may advance more rapidly in one or two commissures while the remaining two or one may be less involved. Thus, a bicuspid transformation of the valve may take place, or but a single slitlike radiating aperture may persist (Fig. 4, *A, B, C, D*). Commissural obliteration (cross fusion of the adjacent free margins of the cusps) beginning at the base of the valve is the most characteristic gross anatomic feature of rheumatic stenosis occurring in the aortic valve (as well as of the mitral and tricuspid).

Late contraction of the accumulated lamellae of scar tissue which have been laid down upon the free margin of the reduced valve orifice may lead to a secondary "purse-string" type of narrowing of the central (or eccentric) residual opening. Thus, the initial stenosis, due to the commissural obliteration, may become even tighter. On the other hand, the major line or direction of placement of the strands of scar tissue applied to the surface of the valve

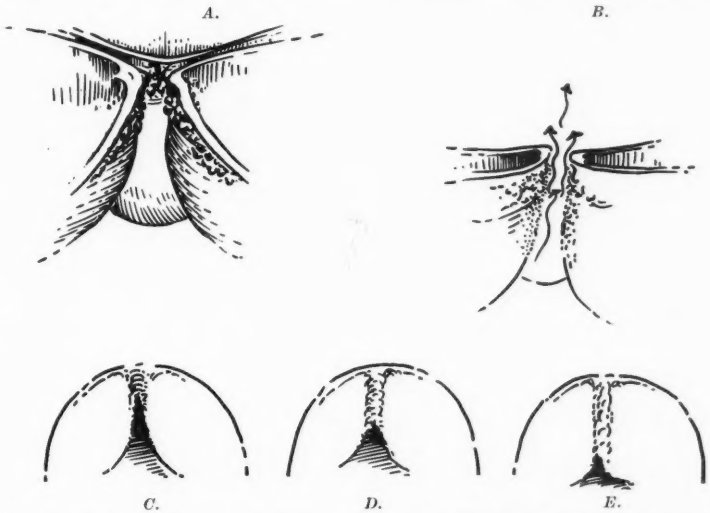


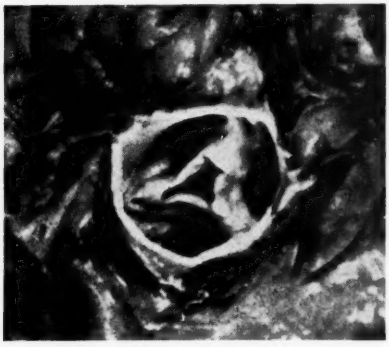
Fig. 3.—*A*, Early appearance of vegetations upon the zones of leaflet approximation in rheumatic aortic valvulitis. This localization may be related to the repeated trauma of valve closure. *B*, The surface irregularities produced by these vegetations engender a turbulence of flow which leads to recurrent deposition of fibrin and platelets upon the ventricular aspect of the valve. *C*, Beginning cross fusion of peripheral portions of adjacent leaflets due to organization of heaped-up fibrin. *D*, Centripetally progressive obliteration of valve commissure. *E*, Final state of nearly complete commissural obliteration. (From Bailey, Surgery of the Heart, courtesy Lea & Febiger.)

may be such that the preponderant late effect is retraction of the thickened margins of the cusps toward their basal attachment. Again, this may be either a symmetrical or an asymmetrical phenomenon. Thus, accurate diastolic coaptation of the apposed valve edges may become impossible, causing a greater or lesser degree of incompetence to become superimposed upon the primarily stenotic lesion. Not infrequently, the clinical evidences of such regurgitation may come to overshadow those of a dynamically less significant stenosis (Fig. 5, *A, B*).

A.

B.

C.



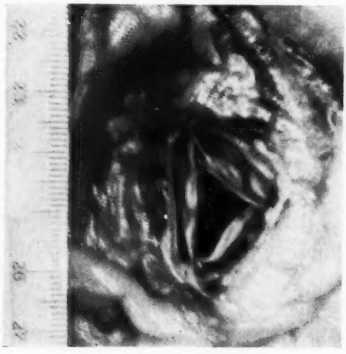
D.

Fig. 4.—*A*, Photograph showing early stage of rheumatic aortic stenosis. Note beginning symmetrical obliteration of the commissures. (Courtesy Dr. Raymond Truex, Hahnemann Hospital, Philadelphia.)

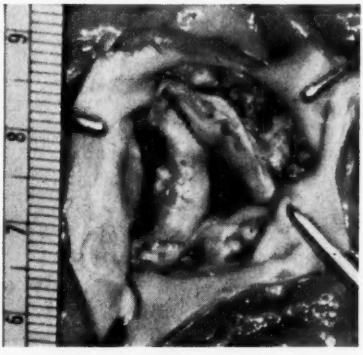
B, Advanced aortic stenosis showing complete obliteration of two commissures, a radiating slitlike opening representing the one remaining unfused commissure. (From McMillan, Aortic Stenosis: A Post-mortem Cinephotographic Study of Valve Action, Brit. Heart J. 17: 56, 1955.)

C, Complete obliteration of one commissure while the others remain open. This results in bicuspid transformation of the valve. (From Bailey, Surgery of the Heart, courtesy Lea & Febiger.)

D, Extensive symmetrical obliteration of all three commissures reduces the valve opening to a diminutive central triangular passage. (From Gould, Pathology of the Heart, courtesy Charles C Thomas.)



A.

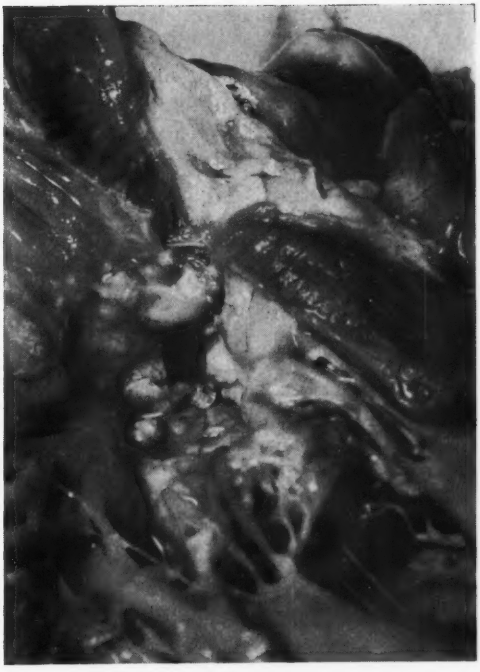


B.

Fig. 5.—*A*, Retraction of central edges of cusps in rheumatic aortic valvulitis brings about an element of aortic insufficiency which sometimes may overshadow the coexisting stenosis. (From Bailey, Surgery of the Heart, courtesy Lea & Febiger.)

B, Aortic regurgitation is often found in association with stenosis when the valve leaflets are extensively calcified. In this case, the insufficiency was not dynamically significant. (From Larzelere and Bailey, Aortic Commissurotomy, J. Thoracic Surg. 26: 66, 1953.)

A.



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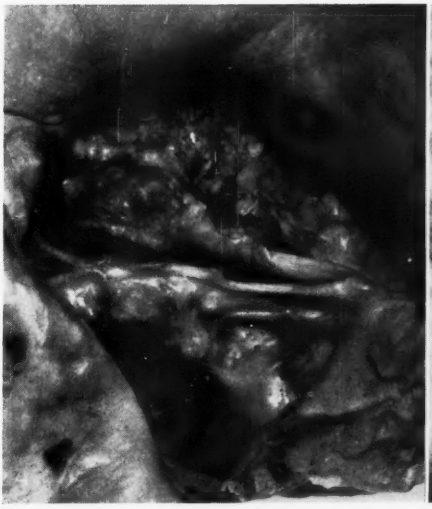


C.

D.

Fig. 6.—*A*, Extensive calcification of the aortic valve seen from the arterial aspect. Note irregular excrescences extending up the wall of the aorta.

B, Ventricular aspect of an extensively calcified aortic valve. This calcification may extend into the septal cusp of the mitral valve as well as into the interventricular septum.

C, "Clamshell-like" aortic valve presenting extreme calcification and only a very tiny slitlike residual orifice (toward the right of the photograph). (From Bailey, Surgery of the Heart, courtesy Lea & Febiger.)

D, Forcible instrumental separation of valve seen in Fig. 6, *C*. Note limitation of possible restitution of valve movement due to extreme rigidity of involved structures. (From Bailey, Surgery of the Heart, courtesy Lea & Febiger.)

With the prolonged immobilization of the thickened leaflets, both due to intrinsic stiffening and to fixation to adjacent cusps, late degenerative changes are prone to develop. This may account, in part, for the very great incidence of calcification of the leaflets observed in aortic stenosis. However, it is probable that the relatively high pressures developed within the left ventricle and aorta and their violent fluctuations with consequent resulting trauma to the valve also are responsible, at least in part, for the greater incidence of calcific

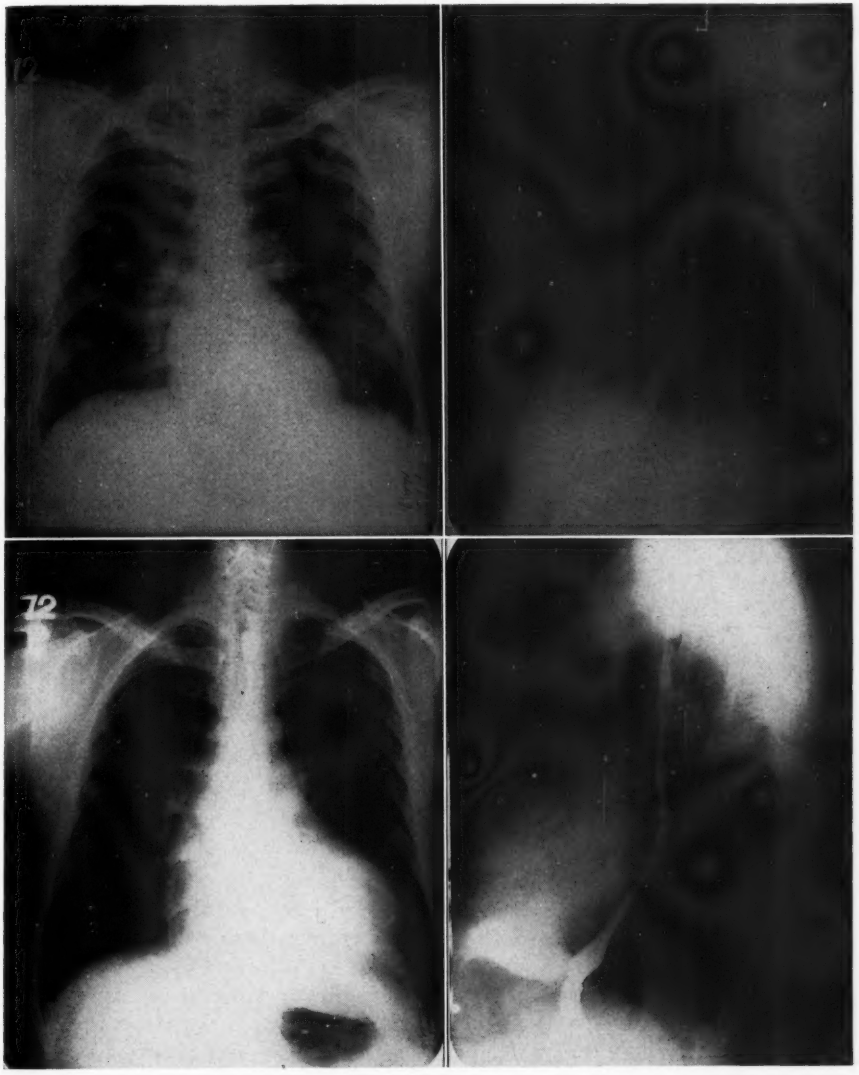
A.

Fig. 7, *A.*—Photograph of autopsy specimen showing extreme thickening of left ventricular wall in aortic stenosis. In pure forms of this lesion, there is no enlargement of the ventricular chamber. (From Bailey, Surgery of the Heart, courtesy Lea & Febiger.)

changes observed in this valve than in others involved by a similar stenotic process. Thus we may explain the relative rarity of calcific changes in stenotic right-sided heart valves (tricuspid and pulmonic) which are subjected essentially to much lower pressures. In mitral stenosis where there is impaired ventricular filling with consequently less trauma to the valve structures, the incidence of calcific changes is of intermediate frequency (38 per cent). Characteristically, calcification of the aortic valve seems to involve the region of the obliterated commissures even more frequently and more extensively than the

margins and fundi of the cusps themselves. Not infrequently, the calcification may extend from the commissures into the adjacent aortic wall, and may encircle it to form a complete calcific ring. Occasionally, the calcification extends up the

B.



C.

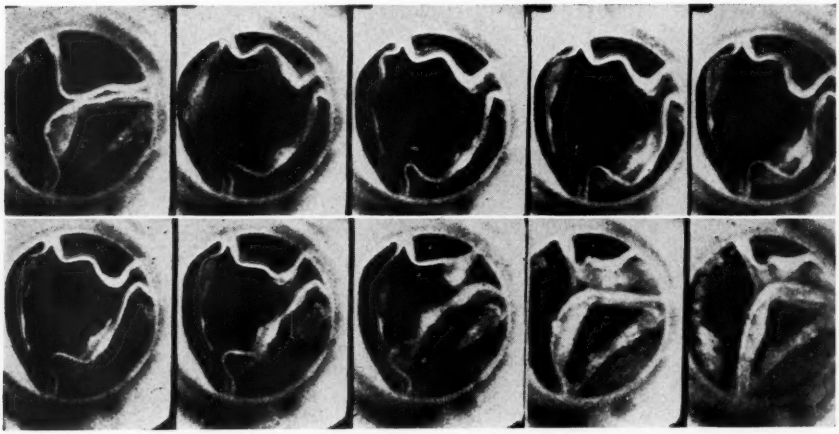
Fig. 7, *B* and *C*.—*B*, Posteroanterior and lateral roentgenograms from a case of pure aortic stenosis. Note relatively small size and normal contour of cardiac silhouette, and the absence of left atrial dilatation. *C*, Comparable roentgenograms from a patient with combined stenosis and insufficiency of the aortic valve. Note larger size of heart and tendency toward "boot-shaped" configuration of contour. The left atrium is not dilated in this case. (From Bailey, Surgery of the Heart, courtesy Lea & Febiger.)

aortic wall in the form of "stalagmites." Sometimes the cusps and adjacent aortic wall become converted into an over-all "volcano-like" or "clamshell-like" structure (Fig. 6, *A, B, C, D*). Characteristically, the sinuses of Valsalva tend to be relatively free of such involvement.

Poststenotic dilatation of the first portion of the ascending aorta is seen not infrequently in long-standing cases of acquired aortic stenosis.

The left ventricle in "pure" aortic stenosis undergoes a "concentric" type of hypertrophy without chamber enlargement. The walls of the ventricle may become as much as 1 inch (2.5 cm.) in thickness (see Fig. 16*C*). However, such concentric hypertrophy may not show on the usual posteroanterior roentgenogram of the heart. When ultimate exhaustion and failure have supervened, dilatation may take place, and the radiographic shadow may assume a bootlike contour. However, dilatation of the chamber usually is the result of a coexisting element of aortic incompetence (Fig. 7, *A, B, C*).

A.



B.

Fig. 8.—*A* and *B*, Successive steps in opening and closing of normal aortic valve as shown by cinephotography. Note that the effective aortic valve aperture during maximal opening has a roughly triangular contour. (From McMillan, Brit. Heart J. 17: 56, 1955.)

The left ventricular wall in aortic stenosis frequently undergoes a peculiar form of degeneration, becoming soft and friable. In some cases, this is so marked that hemostatic sutures placed in the ventricle will not hold securely, but tend gradually to "saw" or cut through the contracting muscular wall. This may result in fatal hemorrhage.

In addition, these ventricles tend to exhibit an exquisite sensitivity or irritability even to relatively slight stimuli. Touching or suturing may set off runs of ventricular extrasystoles. Sometimes, they may progress to ventricular fibrillation.

PATHOPHYSIOLOGY OF AORTIC STENOSIS

As da Vinci[16] postulated in 1513, the effective aortic valve opening during systole is a triangle rather than a circle. That this actually is the situation

recently has been demonstrated by cinephotography by McMillan[17] (Fig. 8, *A* and *B*).

Obviously, the essential effect of these various pathologic changes characteristic of aortic stenosis is to reduce the effective size of the valve aperture and, hence, to create impedance to ventricular ejection. This, in turn, requires an increase in the velocity of flow through the orifice, at least on maximal exertion. As the size of the opening progressively becomes diminished, the required velocity of flow through the orifice becomes greater, and there is an effective prolongation of the "isometric" period of ventricular contraction. As the process continues, ejection is accomplished with more and more difficulty and only by greater and greater ventricular effort.

However, it must be appreciated that these effects usually are not manifest, at least at rest, until the valve opening has become reduced to about 25 per cent of its normal size.[18, 19] Indeed, Gorlin and co-workers[20] have indicated that the critical point of valvular narrowing is at about 10 per cent of normal size. Beyond this point, further reduction in orificial size requires an inordinate increase in ventricular overwork. The period of ejection is prolonged. This is expressed by the development of a large gradient between the intra-aortic and intraventricular pressures during systole. Eventually compensation is no longer possible and the left ventricle fails (Fig. 9).

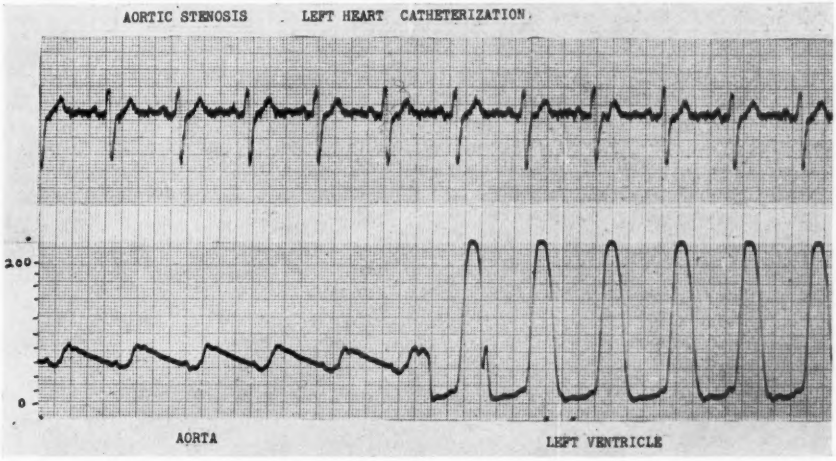


Fig. 9.—Pressure tracing obtained from aorta and left ventricle as catheter is withdrawn during left heart catheterization (transthoracic puncture). Note the large gradient between the respective systolic levels in these two vascular segments.

The altered hemodynamics of flow are expressed grossly (in very severe cases) by changes in the measured blood pressure (low systolic with relatively high diastolic levels), and more intimately by alterations in the brachial arterial pressure tracing (Fig. 10, *A-E*).

The occurrence and position of the anacrotic notch are related to the severity of the stenosis, to the stroke volume, and to the level of the blood pressure.[13, 21] The abnormal relationships are presented most exquisitely by comparative simultaneous evaluation of the intraventricular and intra-aortic

(or brachial arterial) pressure curves. Apparently, they must be performed with the patient unanesthetized and with an intact chest wall since such determinations obtained directly during surgery usually present a significant "physiologic damping" (Fig. 11, *A, B*). Probably this phenomenon is related to a reduction in stroke (and minute) output. For the same reason it is important to measure the cardiac output by simultaneously performing right and left heart catheterization.

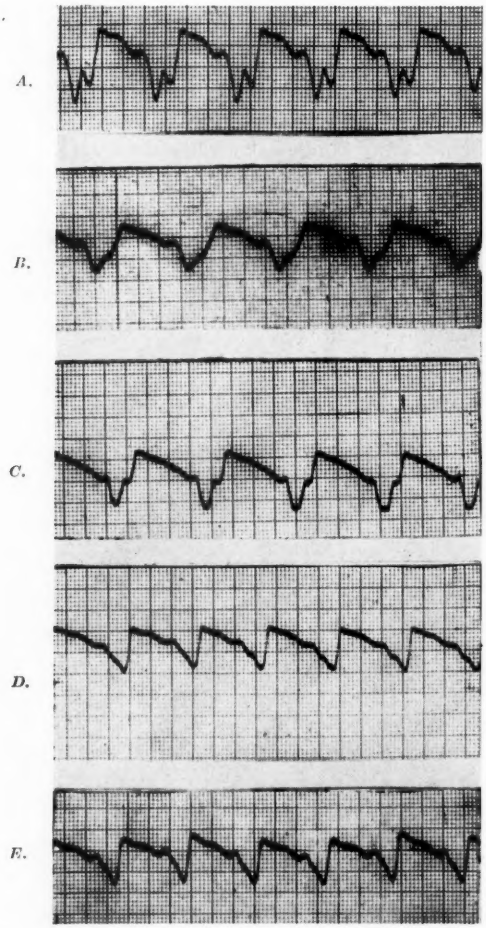


Fig. 10.—*A*, Normal brachial arterial pressure tracing. *B*, *C*, *D*, and *E*, Four patterns of the brachial arterial pressure curves which suggest the existence of a significant degree of aortic stenosis. (From Goldberg, Bakst, and Bailey, The Dynamics of Aortic Valvular Disease, Am. Heart J. 47: 527, 1954.)

Another feature characteristic of severe aortic stenosis is the development of coronary insufficiency. While, undoubtedly, intrinsic coronary arterial disease sometimes may coexist, in most cases the insufficiency is due entirely

to the altered kinetics of flow. Support for this opinion is obtained from the usual finding of anatomically normal coronary arteries at autopsy in individuals who have succumbed from stenotic aortic disease and who have manifested a severe anginal syndrome. Also, it is strengthened by the nearly constantly observed relief of angina, when preoperatively marked, following successful aortic commissurotomy.

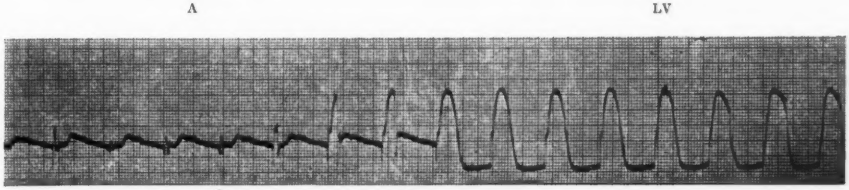


Fig. 11, *A*.

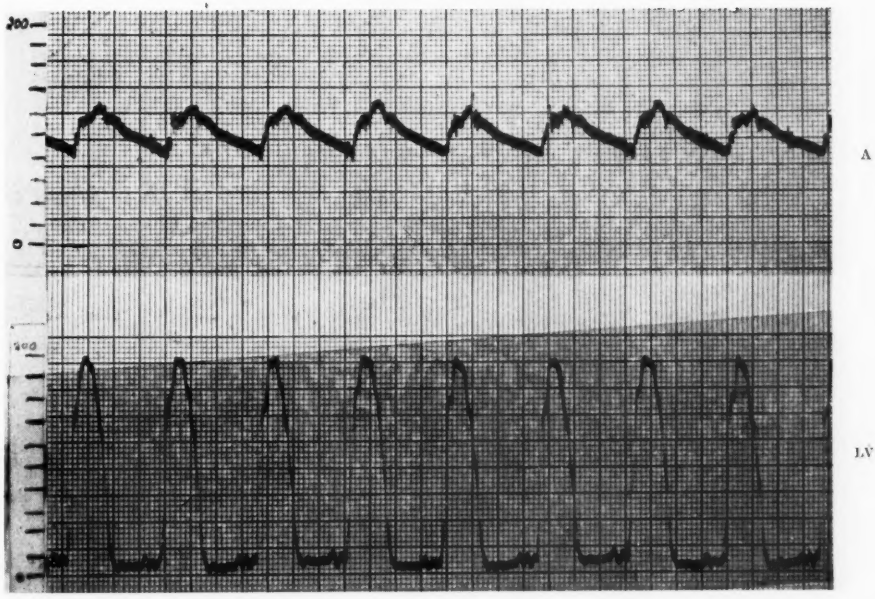


Fig. 11, *B*.

Fig. 11.—*A*, Preoperative aortic (*A*) and left ventricular (*LV*) pressure tracing obtained by catheterization of the left side of the heart (transthoracic approach). The range is 400 mm. Hg, indicating a systolic gradient of approximately 120 mm. Hg. *B*, Simultaneous direct pressure tracings obtained from the aorta (*A*) and left ventricle (*LV*) in the same patient after opening the chest but before definitive maneuvers. Since the range is 200 mm. Hg, the gradient is approximately 70 mm. Hg. Apparently significant alterations in physiology develop during anesthesia and surgery which render impossible accurate correlation with studies performed under other conditions.

It seems probable that the over-all lowering of the pressure at the root of the aorta is associated with a corresponding reduction in coronary flow. Green[22] feels that this reduction occurs largely during systole and is caused by the relatively greatly increased peripheral coronary arterial resistance due

to the prolonged period of high intraventricular pressure (ejection phase). Contratto and Levine[23] have suggested that the high velocity of blood flow (nozzle effect) past the ostia of the coronary arteries during systole might produce a "suction" (Venturi) effect at this point, thus preventing coronary filling during this portion of the cardiac cycle.

Whatever the exact physical explanation, it does appear that coronary filling is impaired in aortic stenosis. Furthermore, since the left ventricle becomes both overworked and hypertrophied in this condition, its demands for coronary flow must be much larger than normal. Hence, a disproportion between coronary supply and demand develops in many of these individuals. This condition was present in 61 per cent of our patients and was indicated clinically by the syndrome of angina pectoris, often initiated by very slight exertion.

CLINICAL COURSE

It appears that for a long time after the beginning of the development of the typical pathologic changes of aortic stenosis the patient presents no clinical symptoms. It is doubtful whether the basal systolic murmur develops until the process has become well advanced. Nevertheless, the patient may remain asymptomatic long after the characteristic short, harsh, basal systolic murmur has become manifest. The onset of definite symptoms usually is followed by a fairly rapid and progressive downhill course without a tendency toward temporary marked remissions following appropriate medical therapy such as is often seen in mitral stenosis.

The earliest symptoms are tiredness and easy fatigability soon followed by exertional dyspnea (85 per cent) and, finally, orthopnea (48 per cent) may develop. All of these symptoms are related to an inadequate cardiac output and left ventricular exhaustion.

Dizziness or frank fainting spells may be reported (65 per cent). They would seem also to be related to an inadequate cardiac output (cerebral anoxia) although it is impossible to rule out the effects of a hyperactive carotid sinus or the sudden onset of an arrhythmia. An anginal syndrome of some type was present in 61 per cent of the patients studied. Electrocardiographic findings were abnormal in 97 per cent of the cases. The most common changes were those indicative of left ventricular hypertrophy and strain. Conduction defects were present in 4 per cent, and when these involved the interatrial bundle, it was difficult to rule out associated coronary artery disease. In 2 of our patients who died following valvular surgery, typical advanced atherosclerotic changes were noted in the coronary arteries. In 1 of them, a fresh myocardial infarct was found. Presumably it was the chief cause of death. While a left ventricular strain pattern was present in 68 per cent of the patients with essentially pure aortic stenosis, in cases of combined aortic and mitral stenosis it was present in less than 20 per cent of the patients.

Patients with aortic stenosis who present a history of dizziness, fainting, or anginal pain have a disturbing tendency toward sudden death. This may occur after very slight exertion. It seems probable that the usual cause is either the sudden onset of acute ventricular fibrillation due to aggravation of severe myocardial ischemia or the persistence of asystole due to circulatory depression following an episode of carotid sinus syncope.

Symptoms related to congestive heart failure may appear. In individuals presenting them prominently, usually the evidences of coronary insufficiency or cerebral anoxia are reciprocally diminished.

PHYSICAL FINDINGS

The most constant and classical abnormal physical finding in aortic stenosis is the short, harsh, basal systolic murmur. When of marked intensity, it often is associated with a systolic thrill. While both are usually maximal over the right second anterior interspace close to the sternal border, either or both of these abnormal signs may be transmitted up the carotid arteries. Sometimes, they may be maximal over the second or third left intercostal space close to the sternal margin. Frequently, the murmur is transmitted to the apex and then may be mistaken for one due to mitral insufficiency. Its shorter length and harsher quality help to distinguish it from the latter which, characteristically, is longer in duration and has more of a blowing quality. Also, the latter tends to replace the first heart sound at the apex.

Generally, the aortic second sound in these patients is weaker than normal and often is absent. However, since it represents the shock of valvular closure, it may be present and of fairly good intensity if an appreciable portion of at least one of the valve leaflets remains flexible enough to provide diastolic competence. When appreciable aortic incompetence coexists, the second sound is usually absent and a long early diastolic blowing murmur, best heard over the left third intercostal space close to the sternum, takes its place. Transmission of this murmur is downward. Such a murmur due to aortic insufficiency occasionally may be heard best at the apex (Austin-Flint), and then may be mistaken for the murmur of mitral stenosis. However, the latter begins later in diastole, tends to be of rumbling rather than blowing quality, and often exhibits a "presystolic accentuation," especially when the cardiac rhythm is regular.

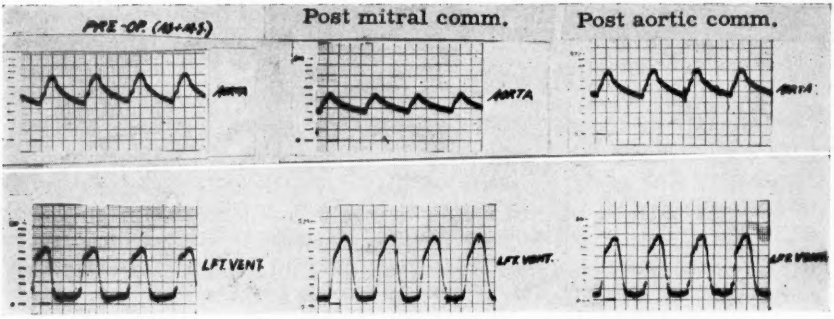
Atrial fibrillation was present in only 3.0 per cent of our patients with essentially "pure" isolated aortic stenosis. Its presence should make one strongly suspect an associated mitral valvular lesion. Atrial fibrillation was observed in 52 per cent of 104 patients with coexisting mitral stenosis and aortic stenosis.

The pulse in advanced aortic stenosis is said to be weak and "plateau-like" and the blood pressure is supposed to exhibit a low systolic level and a relatively high diastolic one. The blood pressure, however, was within normal limits in the majority of our patients.

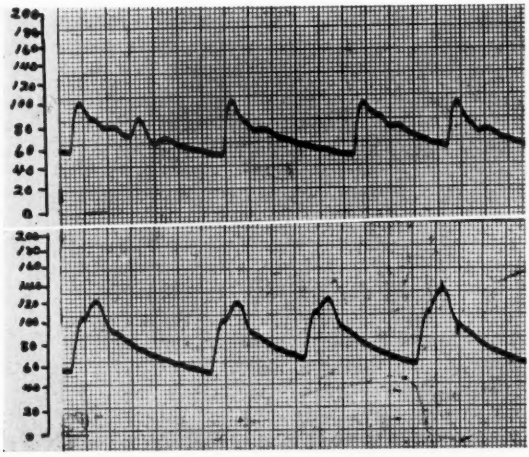
The classical evidences of incipient or clinical congestive heart failure usually are late developments but are of most ominous import.

RADIOLOGIC EXAMINATION

Since the left ventricle in "pure" aortic stenosis undergoes a concentric hypertrophy of its walls without a concomitant enlargement of its chamber, little evidence of enlargement or change may be seen on the posteroanterior projection of the cardiac silhouette (see Fig. 7). However, in advanced stages of the disease with myocardial exhaustion and failure, and especially in the presence of an element of aortic incompetence, both enlargement and a typical



A.



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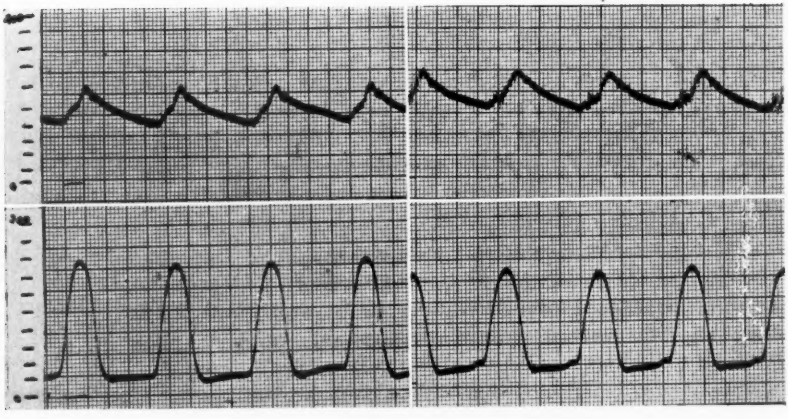

C.

Fig. 12, *A*, *B*, and *C*. (*For legend see opposite page.*)

"boot-like" configuration of the radiographic shadow will be seen. Calcification of the aortic valve is best seen on fluoroscopic examination and was demonstrated in one-half of our cases.

MULTIVALVULAR DISEASE

In patients exhibiting aortic stenosis, either "pure" or combined with a dynamically insignificant degree of aortic insufficiency, one not infrequently finds coexisting mitral valvular disease. Forty-four per cent of our patients of this group were found to have significant mitral stenosis, with or without varying degrees of mitral insufficiency. Five and nine-tenths per cent manifested evidence of mitral insufficiency alone. The majority of this latter group presumably were suffering from dilatation of the mitral annulus fibrosus with resulting functional insufficiency secondary to the left ventricular enlargement caused by the aortic lesion.

In these individuals with combined aortic and mitral valvular disease, one must always consider whether the tricuspid valve may not also be affected. Tricuspid valvular disease was found in 17 per cent of the patients with mitral and aortic rheumatic valvular disease. Predominant tricuspid stenosis was recognized by direct digital examination in 11 per cent of patients with both mitral and aortic valvular disease. It appears that acquired tricuspid stenosis practically never is found as an isolated lesion. It seems always to be accompanied at least by a similar mitral valvular lesion.

Apparently, rheumatic or other acquired stenosis of the pulmonary valve is extremely rare. Acquired pulmonary insufficiency (usually functional) apparently is a lesion of relatively minor clinical significance.

The observed occurrence of significant disease of one or both atrioventricular valves in association with aortic valvular disease is of great importance since failure to relieve all physiologically important lesions may spell failure to obtain clinical improvement or even immediate disaster to the patient. It is a well-accepted hydrodynamic principle that one unrelieved point of serious obstruction in a fluid flow system will impede the passage of the current almost as much as two or more similar points of obstruction, since the limited volume passing through one such barrier can easily traverse the next one.

On the other hand, relief of an early valvular obstruction without simultaneous relief of one farther on in the circulation will have the effect of aggravating the manifestations of this later one. Thus, if mitral stenosis is relieved without concomitant commissurotomy for any significant associated aortic

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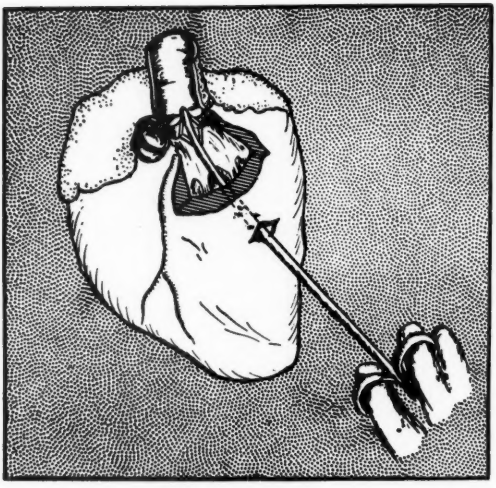
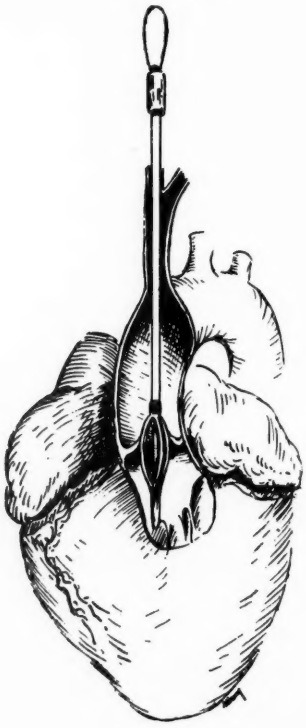
Fig. 12.—*A*, Comparative simultaneous aortic (upper) and left ventricular (lower) pressure determinations obtained at surgery in a patient with combined aortic and mitral stenosis. After relief of the mitral obstruction, the gradient between the systolic levels is increased somewhat and the arterial pressure curve develops the characteristics of aortic stenosis. Both the gradient and the anacrotic slurring are abolished by the immediately ensuing aortic commissurotomy. (From Bailey, Surgery of the Heart, courtesy Lea & Febiger.) *B*, Upper, Brachial arterial pressure curve obtained preoperatively in a patient with mitral stenosis who was considered clinically to have only very mild aortic stenosis. Lower, Brachial tracing taken after mitral commissurotomy showing the typical anacrotic notch of aortic stenosis. Physiologically the aortic stenosis had become dynamically significant because an increased amount of blood was now being expelled through an aortic aperture of unchanged size. *C*, Abolition of systolic pressure gradient obtained between the left ventricle and the aorta in a patient with isolated aortic stenosis. Left, Precommissurotomy tracings. Right, Tracings obtained immediately after aortic commissurotomy.



A.



B.

Fig. 13, *A* and *B*.—*A*, Earliest attempt at surgical division of stenotic aortic value by a modification of Brock's method of pulmonary valvulotomy. The patient did not survive. *B*, Subsequent attempt at dilatation of the stenotic aortic valve by retrograde passage of an instrument with heavy umbrella-spring type blades. The patient survived. (From Larzelere and Bailey, J. Thoracic Surg. 26: 66, 1953.)

stenosis, the clinical importance of the latter becomes enhanced. This is due to the improvement in left ventricular filling which requires this chamber to eject a greater quantity of blood through the same stenotic aortic orifice. Hence, relatively speaking, the aortic stenosis becomes aggravated physiologically. Since isolated aortic stenosis characteristically follows a more malignant course than does the combination of mitral and aortic stenosis, it is evident that such "conservative" treatment must be a disservice to the patient (Fig. 12, *A*, *B*, and *C*).

SURGICAL CONSIDERATIONS

Tuffier[24] in July, 1913, attempted digitally to dilate a stenotic aortic valve in a young male adult by invaginating the dilated anterior aortic wall at a

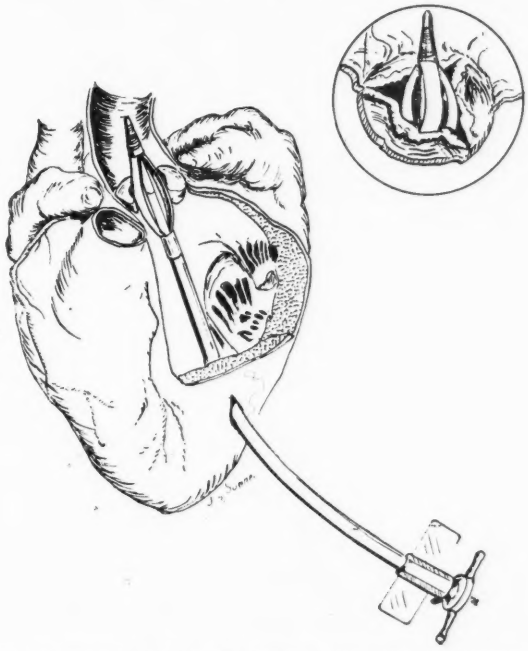


Fig. 13, *C*.—Curved instrument with similar dilator mechanism subsequently used by transventricular approach.

point just above the valve level. The patient survived at least 10 years and was reported improved. Smithy[25] attempted to resect a portion of a stenotic aortic valve in the summer of 1948. He intended to use a modified sphenoid punch inserted through a transventricular approach. Unfortunately, the patient expired suddenly during the induction of anesthesia, apparently due to the onset of acute ventricular fibrillation.

On March 9, 1950, the senior author (C. P. B.)[26] attempted to divide a stenotic aortic valve in a 26-year-old white housewife using a modification of Brock's[27] method of pulmonary valvulotomy and a transventricular ap-

proach. Although the valve was divided according to plan, death on the operating table resulted, due to the creation of a significant degree of insufficiency. This unfortunate experience confirmed certain theoretically-based fears that the sudden production of aortic regurgitation would not be tolerated well in a patient who had become adjusted or "compensated" to a severe degree of aortic stenosis. It was felt, thereafter, that neither cutting across nor excision of a portion of an aortic valve cusp could be justified clinically.

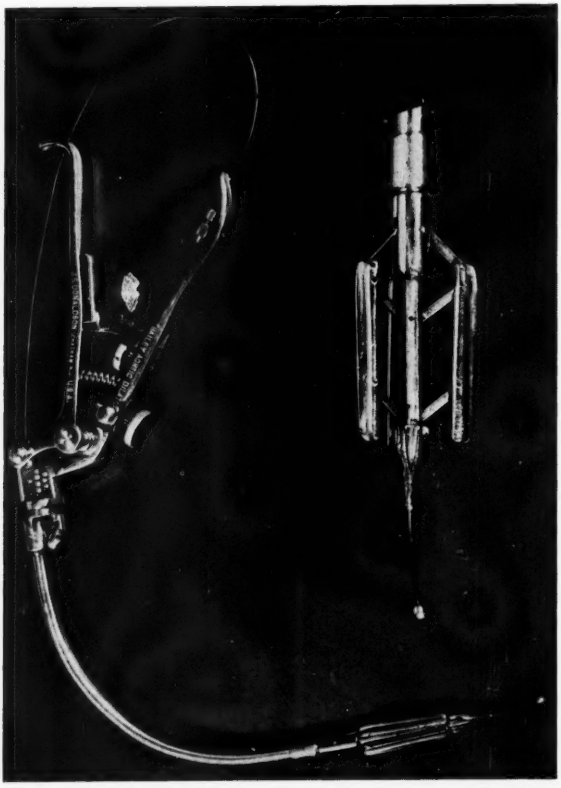


Fig. 14.—Modern triradiate parallel bar type of aortic dilator. Note the incorporated guide wire and olivary tip. The entire head is designed for free rotation on its shaft. This assures accurate automatic alignment of the triangular cross-sectional contour of the open or closed dilator with the outline of the diminutive valve orifice if any remnant of its initial triangularity persists. (From Bailey, Surgery of the Heart, courtesy Lea & Febiger.)

Experimental efforts to "dilate" the stenotic aortic valve in the autopsy room revealed that the fused commissures tended to separate on application of disruptive pressure more readily than did the continuity of the sclerosed leaflets. Hence, it became evident that surgical aortic commissurotomy was a logical possibility. At first, it was felt that this must always be an instrumental accomplishment since it was not then foreseen that deliberate digital examination and manipulation of the valve during life would become feasible.

On April 6, 1950, one of us (C. P. B) performed an instrumental dilatation of a stenotic aortic valve by passing an "umbrella-type" dilator through the valve orifice in retrograde fashion, guiding it through an incision made in the right common carotid artery and through the ascending aorta to the valve orifice which was located by instrumental palpation. While this particular operation was considered successful (with survival), subsequently the procedure was abandoned in favor of a somewhat similar one performed by a transventricular route (Fig. 13, *A, B, C*).

By April, 1952, in large part due to the combined efforts of our engineering associate, Major J. Shearman Donaldson,* and our chief surgical resident, Dr. Henry B. Larzelere,** a much superior instrument was developed for this purpose (Fig. 14). In it, the umbrella-spring type blades have been replaced by three parallel dilating bars with wedgelike cross sections. A retractable guide wire with an olivary tip has been incorporated within the instrument to serve a function comparable to that of the filiform bougie used in the practice of urology. An incorporated rotatory mechanism permits free turning of the dilating head upon its shaft for automatic adjustment of its triangular cross section (open or closed) to the usual residual triangularity of the stenotic

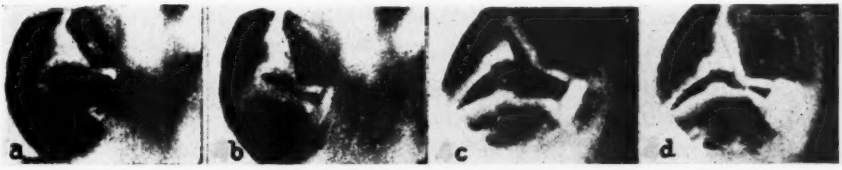


Fig. 15.—*a*, Stenotic aortic valve orifice revealing only one unobliterated commissure—systolic position. *b*, Same valve in diastole (closed). *c*. After passage of triradiate aortic dilator, all three commissures have become separated, and some normal type valve action is provided. Systole. *d*, Closure of valve indicates that competence is fully maintained. (From McMillan, Brit. Heart J. 17: 56, 1955.)

valve orifice. Thus, the unsharp edges of the dilating bars tend to become aligned accurately against the respective angles of the valve orifice. Since these angles represent the central extremities of the fused commissures, mechanical separation of the bars applies parting or splitting pressure directly and most effectively against the commissures, thus tending to separate and mobilize the three functional valve elements anatomically. It was realized that in many valves extreme and perhaps asymmetric sclerosis or calcification might prevent complete separation of all commissures, and that indeed it might be accomplished only partially, perhaps in only one or two of the three. However, it was reasoned that any measure of anatomic separation accomplished would represent at least a partial restoration of a more normal valvular state and would result in a degree of functional improvement. To obtain the desired effect, it was deemed essential that at least some valvular or aortic wall flexibility remain. Since the left ventricle would, in these very cases, be maximally adjusted to compensate for any residual unrelieved aortic stenosis, enlargement of the orifice to more than one-quarter of its normal size would result

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**Dr. Henry B. Larzelere, Toledo, Ohio.

essentially in full clinical relief. This concept depended for its validity upon the maintenance of a state of valvular competence at least the equal of that existing preoperatively (Fig. 15, *A, B, C, D*).

In large measure, these concepts and hopes have been justified by our subsequent clinical experiences. On April 4, 1952, the improved instrument was used with great initial success (by the transventricular route) in a male patient who was nearly moribund. He attained an excellent state of improved health and maintained it for two years subsequently. Ultimate recurrence of symptoms with an unusually rapid downhill course precluded a second surgical attempt. Since then, 211 patients with prominent evidence of aortic stenosis have been operated upon by this instrumental technique, most of them obtaining excellent clinical and physiologic improvement. When mitral stenosis has coexisted, as it did in over 40 per cent, mitral commissurotomy was carried out simultaneously by way of the usual left appendageal approach. However, the operative mortality (Table I) associated with this technique has remained relatively high.[13, 28, 29] Daniel and Scott,[30] Muller and Hyman,[31] and McMillan[17] have reported similar experiences. In order to eliminate nonrelevant considerations such as those related to the presence of a major element of aortic or mitral insufficiency, or of coexisting tricuspid disease, we have tabulated the operative results only in those patients considered to have preponderant aortic stenosis and those with combined preponderant aortic and mitral stenosis (Tables I and II).

TABLE I. TRANSVENTRICULAR APPROACH FOR AORTIC COMMISSUROTOMY SHOWING EARLY OPERATIVE RESULTS

TYPE OF LESION	NUMBER OF CASES	NUMBER OF OPERATIVE DEATHS	OPERATIVE MORTALITY (%)
Aortic stenosis	37	9	24.2
Aortic stenosis with insignificant aortic insufficiency	31	10	32
Total	68	19	28

TABLE II. OPERATIVE RESULTS IN COMBINED SIMULTANEOUS AORTIC AND MITRAL COMMISSUROTOMY IN PATIENTS WITH AORTIC AND MITRAL VALVULAR DISEASE (TRANSVENTRICULAR AND LEFT AURICULAR APPROACHES)

TYPE OF LESION	NUMBER OF CASES	NUMBER OF OPERATIVE DEATHS	MORTALITY (%)
Aortic stenosis plus mitral stenosis	22	1	4.5
Aortic stenosis plus mitral stenosis with mitral insufficiency	10	2	20.0
Aortic stenosis with aortic insufficiency, plus mitral stenosis	24	4	16.7
Aortic stenosis with aortic insufficiency, mitral stenosis with mitral insufficiency	31	9	29.0
Total	87	16	18.4

Consideration of the various ways by which this risk might be reduced has led to some interesting speculations. Breaking down the over-all series of cases into pertinent smaller groups (Tables I and II) revealed that patients with com-

bined aortic and mitral stenotic lesions simultaneously corrected apparently ran less risk than those with essentially "pure" isolated aortic stenosis in whom the aortic valve alone was operated upon. Apparently, there is a dynamic difference in the way isolated aortic stenosis and combined aortic and mitral stenosis affect the heart and circulation.

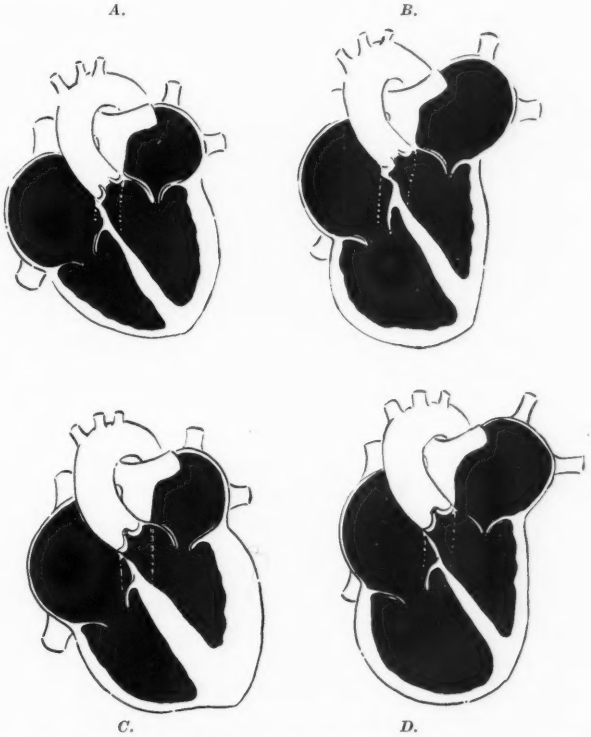


Fig. 16.—Diagrammatic representation of respective types of chamber involvement with various types of valvular stenosis. *A*, Normal heart. *B*, Heart in isolated mitral stenosis. Note the dilated right ventricle and left atrium and the small left ventricle—this latter chamber is protected by the inlet stenosis against overwork or strain. *C*, Heart in isolated aortic stenosis. Note the tremendous concentric type of hypertrophy of the left ventricle without dilatation of the chamber resulting from the overwork consequent to the severe outlet stenosis in the presence of normal filling (no inlet stenosis). The left atrium and right ventricle remain normal until left ventricular failure is imminent. *D*, Heart in combined mitral and aortic stenosis. Note similarity to condition found in isolated mitral stenosis. The right ventricle and left atrium become dilated and overworked but the left ventricle remains small and relatively healthy. It is protected against the effects of outlet obstruction by the severe inlet, stenosis. The small amount of blood entering the left ventricle is readily expelled through the reduced aortic outlet.

Fig. 16 indicates the different types of chamber involvement seen in these various states of aortic or mitral stenotic valvular disease. It is evident that in isolated aortic stenosis the left ventricle bears the full brunt of the disease, becoming overworked and hypertrophied due to its outlet obstruction. As has been mentioned previously, it has been observed that the left ventricular wall in this condition tends to become soft and degenerated, and hence to hold

sutures with difficulty. This tendency for sutures to cut through the soft myocardium was responsible for at least two deaths from hemorrhage, as indicated in Table III.

TABLE III. CAUSES OF DEATH IN PATIENTS OPERATED UPON FOR AORTIC STENOSIS BY TRANSVENTRICULAR TECHNIQUE

CAUSE OF DEATH	NUMBER OF DEATHS
Failure, unclassified	1
Cardiac arrest	5
Ventricular fibrillation	5
Hemorrhage, uncontrolled	2
Cerebral embolism	3
Hypotension (after leaving operating room)	1
Hypotension (in operating room)	1
Cardiac arrest Ventricular fibrillation Laceration of coronary artery	1
Total	19

Furthermore, the hypertrophied left ventricle in these patients very often exhibits hyperirritability tending on relatively slight stimulation to develop runs of ventricular extrasystoles or even ventricular fibrillation. A number of deaths which occurred in the operating room were due to the latter arrhythmia (Table III). Cardiac asystole, perhaps related to the myocardial degeneration, also was a prominent cause of death in this series.

In contrast, the left ventricle in patients with combined aortic and mitral stenosis (see Fig. 16, *D*) characteristically is small and of firm structure readily capable of holding hemostatic sutures securely. It exhibits little or no evidence of unusual irritability to mechanical stimulus. In general, at operation the heart in combined aortic and mitral stenosis assumes the configuration of that seen in pure mitral stenosis, presenting a large right ventricle, pulmonary artery, and left atrium, with a normal-sized or small left ventricle. Of course, it is understood that varying intermediate proportions of these effects may be observed with various degrees and combinations of these lesions.

Post-mortem examination in several of the operated patients showed that a degree of aortic insufficiency inadvertently had been produced during the surgical procedure. In at least two instances, a valve orifice which had developed a bicuspid configuration had resisted tricommissural separation with resulting laceration of the anatomic continuity of a sclerotic cusp. In another, an indescribably distorted calcareous valve had become irregularly fractured, probably resulting in insufficiency. Likoff and associates[29] have reported a 13 per cent incidence of clinically increased aortic insufficiency in patients surviving instrumental transventricular aortic commissurotomy. Varying degrees of regurgitation were created in 5 per cent of the cases without detectable insufficiency prior to surgery.

Because of these anatomic variations, it seems probable that there will always remain a certain irreducible incidence of created aortic regurgitation in purely instrumental attempts at tricommissural separation of the stenotic aortic valve. How much influence such inadvertent creation of insufficiency

had upon those deaths listed under the terms "cardiac arrest," "hypotension," "renal failure," "heart failure," and "ventricular fibrillation" cannot be estimated with accuracy. However, deaths from such causes have become significantly rare since the development and adoption of the now preferred transaortic digitally guided technique of performing aortic commissurotomy.

After due consideration and reflection, it was concluded that, at least in cases of isolated aortic stenosis, the development of some approach to the stenotic aortic valve other than the transventricular one was mandatory. It was deemed of paramount importance that direct digital examination and guidance of instrumentation be incorporated into the procedure if at all possible. McMillan[17] recently has voiced a similar hope. Since both Brock[32] and the senior author (C. P. B.)[26] already had had previous bitter experiences in attempting to approach the aortic valve by way of the left atrium and the mitral orifice, our attention was directed toward the ascending aorta as a possible avenue of approach.

Experiences with direct incision of the aortic wall in the experimental laboratory regardless of the use of various hemostatic devices convinced us that a better means of entrance into its lumen must be devised. Vascular homograft segments sutured to the aortic wall proved unsatisfactory largely because of the fragility of their structure. However, the use of pericardium to form a pouch or "appendage" upon the aorta proved reasonably satisfactory, and gradually a suitable technique for its application was developed. For the first time, it became possible methodically to palpate the aortic valve and to manipulate it under the guidance of "digital vision" without any greater difficulty or disturbance to the cardiac action than is experienced in comparable exploration and manipulation of the mitral valve.

On April 4, 1953, the first successful digitally guided aortic commissurotomy by the supravalvular route was performed by one of us (C. P. B.), using a pouch prepared from the patient's own pericardium. On that occasion, valvular dilatation was accomplished by inserting the three-bladed aortic dilator through a second opening made in the pouch and guiding it through the valve with the intravascular finger.[33] Since that time 61 patients have been operated upon by this method. In 40 per cent of the cases, a satisfactory commissurotomy has been accomplished by digital pressure only. In the remainder, instrumental assistance has been required.

A two-bladed dilating instrument has been designed* to be worn on the finger and inserted with it through the pouch and into the aorta (Fig. 17). Its two nonsharp wedgelike blades separate at a 110-degree angle to part simultaneously those two commissures which, at that time, the intra-aortic finger is able to feel. By the sense of touch, the finger readily can follow the splitting effect upon the valve, and so is able to limit or control it.

We have tried latex, fabric, and plastic pouches, as first suggested by Glenn[34] and later by Swann,[35] but still prefer pericardium for this purpose. Pericardial homografts may be prepared and preserved by the method of

*Obtainable through George P. Pilling & Son Company, Philadelphia, Pa.

Meeker and Gross.[36] Their use obviates the necessity for removing most of the patient's own natural pericardial support, and also for transecting the sternum and opening both pleural cavities.

As an alternative method, plastic fabric may be used to fashion an aortic pouch. Of the various materials of this type we have tried, Vinyon fabric* has proved most satisfactory, mainly because of its elasticity and lack of tendency to fraying. However, a square of nylon cloth may be used provided the cut edges (peripheral as well as about the central incision) are flashed momentarily through the flame of a lighted match. The heat melts the plastic slightly and seals the cut edges. With both fabrics we have found a technique similar to that employed with the pericardial homografts reasonably satisfactory.

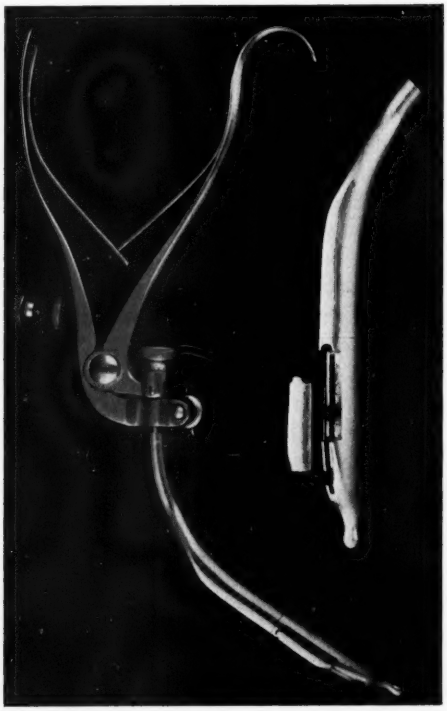


Fig. 17.—Two-bladed aortic dilator designed to be worn on the operator's finger and inserted with it through an artificially created aortic pouch sutured to the artery to permit an approach to the valve in retrograde fashion. (From Bailey, Surgery of the Heart, courtesy Lea & Febiger.)

At the suggestion of one of the authors (H. E. B.), this operation has been modified so that it can be carried out satisfactorily through a right anterior intercostal incision only (third interspace). With one intact pleural space (left) and an undivided sternum, an effective cough mechanism is maintained, and thus the postoperative problems of evacuating the bronchial secretions are minimized.

*Obtainable from American Viscose Corp., Marcus Hook, Pa.

At first, it was not deemed feasible to operate simultaneously for a coexistent mitral stenosis either from the right side or by way of a left anterior intercostal approach, with the patient placed in the supine position preferred for transaortic commissurotomy. Therefore, in view of the observed lower operative risk with coexistent aortic and mitral stenosis (Table II), it was advised formerly that the transventricular route to the aortic valve be chosen in patients with combined mitral and aortic stenosis.[13, 28, 33] Since the lateral posture usually was employed for the aortic dilatation, the mitral valve was readily approached by way of the left auricular appendage either just before or just following the definitive aortic surgery.

However, certain unsatisfactory operative results obtained in such combined cases, and the development of a satisfactory approach to the left atrium and mitral valve by way of the dissected interatrial groove[13, 37] have caused the authors to alter this expressed opinion.

Indeed, the experience and satisfaction obtained in operating from the right side for mitral stenosis combined with aortic stenosis have since led us to choose this approach electively in many patients with isolated mitral stenosis. Actually, in 50 of the last 62 patients operated upon, with a preoperative diagnosis of mitral stenosis alone, at our Clinic have been operated upon from the right side. The chief apparent advantages of the right-sided approach to the stenotic mitral valve may be summarized thus:

1. It is feasible routinely to explore the tricuspid valve by way of the right auricular appendage and to perform, if necessary, tricuspid commissurotomy for stenosis, or surgical plication[38] of the mural portion of the tricuspid annulus fibrosus for incompetence.

2. It is feasible routinely to palpate the external surface of the root of the aorta and the left ventricular outflow tract (and the septal leaflet of the mitral valve) in order to recognize the presence of aortic valvular disease, and directly to attack this lesion immediately, if deemed advisable. In certain of the patients, the aortic cusps may be examined digitally by passing the intracardiac finger through the mitral valve orifice and up to the aorta.

3. Ability routinely to explore the interatrial septum from the right (by way of the right auricular appendage) and to carry out efficiently and immediately an atrio-septo-pexy procedure if an interatrial septal defect is found. Previously unsuspected interatrial septal defects were found in 2 of the patients so explored during the course of a mitral commissurotomy from the right side and were promptly corrected.

4. Ability to approach the mitral valve without disturbing either fresh or old clotted material which is found in approximately 25 per cent[13, 38] of all patients operated upon for mitral stenosis. Since fragmentation of such clots is the chief source of operatively produced arterial embolization, a serious complication which has occurred in 6.1 per cent[13] of our entire series of mitral commissurotomy operations performed from the left-sided approach (1,500), it would seem that avoidance of this complication should be an operative objective of primary concern to the surgeon. Such clots extend well into the true atrial chamber in 13.6 per cent of those affected,[13] sometimes coming to occupy 25 to 30 per cent of its entire luminal capacity. Apparently (unlike fibromyxomatous

interatrial tumors) they never are found attached directly to the interatrial septum. Hence, a right-sided entrance into the left atrium by way of the interatrial groove would seem "always" to avoid disturbing such a thrombus.

5. Technically, a superior type of mitral commissurotomy may be accomplished from the right side. In large part, this is related to the relative ease and safety experienced in separating the posteromedial mitral commissure, a maneuver often fraught with difficulty and danger and hence usually deleted whenever possible by most operators, when mitral commissurotomy is carried out through the usual left-sided approach.

6. The presence of a nearly virgin operative field in a patient who previously has been subjected to commissurotomy from the left side. This problem will be encountered more frequently in the future as mitral commissurotomy is undertaken more often at an increasing number of clinics. Since, in most instances, the left auricular appendage will have been obliterated surgically, this advantageous "tunnel" of entrance[38] from the left usually will have been destroyed.

7. Extensive calcification of an old laminated thrombus lining the left lateral aspect of the left atrium (a relatively rare complication) in the presence of small pulmonary veins may require the use of the right-sided operative approach.

8. The supine operative position employed for the right-sided approach physiologically is easy on patients with borderline circulatory stability. Many of these patients cannot tolerate the lateral position which is preferred for the performance of mitral commissurotomy from the left side. They may develop severe hypotension as soon as they are turned into the lateral operative position.

9. In patients operated upon by the right-sided approach, obviously there is no danger of laceration of the left auricular appendage. This is of significance only in that the respective appendages are the thinnest, the most fragile, and, hence, the most easily lacerated portions of the entire cardiac wall.

10. The atrial attachment of a transvalvular pericardiocartilaginous "stent" used for the correction of mitral regurgitation of the posterior "teardrop" variety is best placed in the region of the right interatrial groove in order to ensure the persistent accurate application of the wider extremity of the cross-sectional contour of the stent into the rounded incompetent posterior "commissure."

11. In the presence of a persistent and recurrent right-sided pleural effusion (from heart failure), the right-sided approach permits one to avoid surgically collapsing the functional left lung, and at the same time to correct the pleuropulmonary condition by lung decortication.

12. Postoperatively, there is less incisional pain and delay in re-establishing arm function with the right-sided anterior thoracic wound.

13. A more esthetic operative scar and a less crippling operative incision result from the right-sided approach. Since the right anterior incision is placed in the inframammary groove in the female, it readily may be hidden by the usual brassière type of breast support (see Fig. 26, *B*). The patient, often a young woman, then may wear a low-backed or low-necked gown without psychologic stress. In the male, the crippling effect of splitting and surgical

division of a relatively few fibers of the right pectoral muscles is insignificant in comparison with the great muscle damage produced by interruption of the continuity of major portions of the rhomboideus, latissimus dorsi, serratus, and (sometimes) erector spinae muscle groups (see Fig. 26, *C*).

On the other hand, the disadvantages associated with the right-sided approach to the mitral valve seem to be:

1. Somewhat less than the usual facility will be experienced in separating the fused anterior mitral valve commissure from the right. This is related largely to an inability to apply appropriate external counter pressure over the commissure. Hence, an instrumental division of this commissure is required more frequently than when the left-sided route is used.

2. One experiences considerable difficulty in dissecting up the innominate and left common carotid arteries from the right-sided incision in an effort to diminish the incidence of cerebral embolization during the course of surgery. As mentioned, this disadvantage largely is nullified in our opinion by reason of the avoidance of direct manipulation of a possible thrombotic appendage when the valve is approached from the right.

3. One cannot plicate the mural portion of the fibrous ring of the mitral valve in order to correct mitral regurgitation of the annular type from the right side.

4. Obviously, the left auricular appendage cannot be ligated through a right-sided approach. Hence, one is unable to ensure that arterial embolization at a later date may not derive from thrombosis of the unobliterated left auricular appendage. We feel that adequate enlargement of the mitral orifice offers a reasonably reliable guarantee against further atrial thrombosis, and, hence, against arterial embolism as indicated by the study of Storer and associates.[46]

5. Inability to massage the ventricles properly through the right-sided incision in case of cardiac arrest. Such an accident would demand prompt opening of the left hemithorax and usually transection of the sternum.

6. There is always a possibility of losing hemostatic control of the atrial incision due to the unexpected detachment, breaking, or inadvertent cutting of the constricting purse-string suture placed about the dissected portion of the interatrial groove. Simple approximation of the two lips of the dissected interatrial groove with rat-toothed thumb forceps or Allis clamps will stop this type of bleeding.

7. A freely floating or "ball-type" of intra-atrial thrombus is more difficult to remove from the left atrium by the right-sided approach.

8. A surgeon with a short index finger may experience difficulty in separating the anterior mitral commissure in a patient with a very large left or dilated atrium.

When all is considered, we have come to the conclusion that since rheumatic valvulitis tends to present multiple sites rather than a single site of valvular involvement and since clinical and physiologic investigations in their present state of development are rather crude and often unreliable, it is better to explore digitally (only by external palpation of the aortic valve, however) and to treat definitively and immediately, if necessary, all three valves which

may be subject to such involvement in nearly every case operated upon for any one such lesion. It is obvious in order to implement this concept that the right-sided approach should be chosen.

Today, stenotic lesions of both the aortic and mitral valves readily are correctible through an incision made in the right third intercostal space. Furthermore, the possible presence of tricuspid stenosis which unfortunately appears often to be undiagnosable by the usual methods of preoperative investigation, readily may be determined with certainty by direct digital palpation using the same thoracic incision.

We now feel that every case of acquired aortic stenosis, with or without evident mitral involvement, of a severity sufficient to justify surgery should be operated upon by way of an anterior right third intercostal incision using a transaortic approach to the valve. Since the right auricular appendage lies over the first portion of the aorta, it may tend to "get in the way" of the operator. Therefore, we prefer to explore the tricuspid valve digitally by way of the right auricular appendage before directing our efforts toward the aortic lesion. If tricuspid stenosis is found to coexist, it may be relieved either by digital pressure or by digitally guided instrumental means. Then, the appendage is amputated partially. It is ligated at its base, and is oversewn.

After the aortic commissurotomy has been completed, thought is given to the advisability of exploration of the mitral valve. If the clinical features of the case suggest combined valvular involvement (especially the presence of atrial fibrillation), or if the tricuspid valve has been found to be stenotic (apparently this is never found in the absence of coexistent mitral stenosis), the mitral valve should be explored digitally by way of the dissected interatrial groove. Indeed, if the patient's condition is not precarious after the completion of the aortic surgery, it is the authors' custom routinely to explore the mitral valve in order at least to rule out a possible unsuspected obstruction. If stenosis is found, an appropriate digital or instrumental mitral commissurotomy is carried out from the right side.

Coexisting mitral insufficiency, in a patient with aortic stenosis, is usually of a functional nature and relatively seldom requires definitive treatment. When it does, the procedure of applying a transvalvular pericardiocartilaginous stent as described elsewhere[38] may be undertaken.

Selection of Patients.—In general, it is felt, as in selecting patients for mitral commissurotomy, that all patients with aortic stenosis who might be considered as belonging in functional Class II or III (New York Heart Classification) may and should undergo aortic commissurotomy using the supravalvular approach. It has been our policy to operate upon Class IV patients whenever their condition can be brought to such a point that it is felt they logically might be expected to survive surgery. Whether this attitude will prove to be unduly optimistic, only time will tell.

In the past, we have leaned heavily upon the brachial arterial pressure curve in patients who presented somewhat questionable indications for surgery. While we fully realize the shortcomings of this method of evaluation, it does offer an additional and an essentially safe method of evaluating the functional importance of the aortic narrowing.

The ideal method of mensurating the degree of existing aortic stenosis, of course, is left heart catheterization, and this study is now applied routinely in all of our patients who are suspected of having aortic stenosis. This may be carried out either by puncturing the left atrium by the use of a long needle inserted through a bronchoscope[39] or by inserting one through the intact thoracic wall (the right eighth intercostal space).[40, 41] We have always used the latter method. A polythene catheter is passed through the needle to enter the left atrium. Then, an attempt is made to cause it to traverse the mitral valve to enter the left ventricle. In some cases (15 of 50; or 30 per cent), it may even be passed upward and into the aorta. If this latter maneuver proves to be impossible, a simultaneously obtained brachial arterial tracing will afford a reasonably comparable pressure curve for the determination of any gradient which may exist between the ventricle and the aorta (see Fig. 27, *A* and *B*). Whenever possible, left heart catheterization should be performed simultaneously with a right heart catheterization. Thus a complete physiologic study and the relation of the gradients to the cardiac output may be established.

At the same time, if any exists, a left atrioventricular gradient indicative of mitral stenosis may be detected. We do not advise aortic commissurotomy unless a transaortic gradient of 40 mm. Hg or greater can be established.

Unfortunately, this desirable scientific study is not feasible in all patients. We have only been able preoperatively to pass the catheter from the left atrium through the mitral valve and into the left ventricle in 88 per cent of our studied patients. Furthermore, because of the inherent risks associated with the method (possibility of intrapericardial hemorrhage, arrhythmia, pneumothorax, infection), it probably will never be applied generally in many clinics in the evaluation of these patients.

In children with congenital stenosis of the aortic valve, surgery is advised because of the high mortality associated with conservative management (Downing[42]). Here the approach chosen usually will be the transventricular one because the small aortic lumen may not admit the finger.

Contraindications.—It is felt that most of the pertinent contraindications to surgery for aortic stenosis are the natural common sense ones. Certainly, no patient should be subjected to aortic surgery if he is in a terminal or unprepared state. Neither should he be operated upon when in a phase of acute rheumatic activity or during the course of active subacute bacterial endocarditis.

Coexisting significant regurgitant lesions of the mitral or aortic valves are severe deterrents to aortic commissurotomy. However, we frequently accept such patients on a calculated risk basis if we feel that at the same time we will be able definitively to alleviate the regurgitation, or if we feel that the patient will receive some net gain from correction of the stenotic lesion alone.

The finding of a very large heart is suggestive of complete myocardial exhaustion, and implies an inability of the myocardium to respond favorably even after proper relief of the obstruction. Nevertheless, some clinically dramatic results have been obtained in occasional selected patients with very large hearts.

Extreme calcification of the valve recognized during fluoroscopy does not constitute a contraindication to an attempt at surgery, although it sometimes does nullify the effectiveness of our effort.

Preparation for Surgery.—The steps in the preparation of a patient for aortic commissurotomy are very similar to those employed in mitral surgery. In general, digitalis, diuretics, salt restriction, ion exchange resins, and rest therapy are appropriate whenever incipient or actual failure presents. Likoff and associates[29] feel strongly that the patient's blood volume must be brought down to normal limits before final surgical consideration is undertaken. Exhibition of adequate therapeutic doses of all the usual vitamins, especially thiamine, is in order for several days preoperatively to overcome any possibly unrecognized subclinical vitamin deficiency.

In patients who have been receiving large doses of cortisone, adrenal function may be depressed. In such individuals, both cortisone and aqueous cortical adrenal extract are given for several days before surgery, and this therapy is maintained postoperatively for a week or more.

SURGICAL TECHNIQUE

Transventricular Instrumental Commissurotomy.—For the reasons previously outlined we have come essentially to limit the transventricular technique for the surgical relief of aortic stenosis to the treatment of two types of patients: (1) children with congenital aortic stenosis (either valvular or subvalvular), and (2) adults in whom mitral surgery is being performed from the left side, and who are found at operation (for the first time) to have a previously unsuspected significant degree of aortic stenosis. This latter condition is suggested by the palpation of a marked systolic thrill over the root of the aorta after the relief of the mitral obstruction. The decision to intervene definitively is made by measuring a significant systolic pressure differential (over 25 mm. Hg) between the aorta and left ventricle by direct insertion of a needle into each chamber. Even better, these measurements may be taken before and after relief of the mitral obstruction to detect any increase in the physiologic impediment produced by the relief of the ventricular inlet obstruction (see Fig. 12).

In children the transventricular approach perforce is chosen because the small size of the aortic lumen usually is not adequate to admit a finger for exploration. By limiting the extent of possible expansion of the dilator blades a serious laceration of the usually funicular congenitally deformed valve structure will be avoided. However, the supravalvular approach obviously would be preferable in the accomplishment of separation of a bicuspid type of valve. The child's ventricle tolerates this type of intervention unusually well as long as no great amount of insufficiency is produced (no deaths in 9 such procedures).

When the transventricular approach is chosen electively for aortic commissurotomy, the patient usually is placed laterally with the left side uppermost. The chest wall is incised in the usual curved posterolateral or peri-

scapular thoracic fashion. The muscles of the chest wall are divided in the line of the skin incision. Bleeding points are clamped and either ligated or coagulated with the high frequency current. When the anterolateral approach is chosen the incision is made in the left inframammary groove but is extended somewhat posteriorly across the axilla. In all cases the left fifth intercostal space is opened and the ribs are spread widely with fixed rib spreaders (Finochietto or Burford type).

The lung is retracted posteriorly and the pericardium is incised, preferably for a limited distance only. This incision should expose the apical 5 cm. of the left ventricle, and is made anterior and parallel to the left phrenic nerve. A second longitudinal incision is made in the pericardium at the base of the heart, so that the fingers and thumb of the operator's left hand may be inserted to encircle the root of the aorta. The margins of the pericardial incisions may be held apart by weighted sutures.

An "avascular" area is selected upon the ventricular surface, usually centered 3 cm. from the apex, and 3 cm. lateral to the anterior descending coronary artery. A purse-string suture of heavy silk or nylon is placed deeply into the ventricular substance in "triangulation" fashion (three bites into the myocardium with three intervening superficial loops). The ends of the suture are incorporated within a Rumel-Belmont flexible tourniquet. The circumscribed region should be about 2 cm. across.

A tiny puncture is made down to the lumen in the center of this outlined area with a slender bistoury type blade. The olivary tip of the guide wire is drawn out about 12 cm. from the tapering tip of the triradiate aortic dilator. It is inserted through the puncture wound into the ventricular chamber, and is directed along the interventricular septum and up toward the aortic valve. Usually it will be guided by the funicular valve to its orifice, and will traverse this diminutive opening without difficulty, entering the supravalvular portion of the aorta. The fingers of the operator's left hand, palpating the posterior aspect of the ascending aorta usually will be able to detect the olivary tip pressing against the posterior aortic wall and perhaps causing it to protrude in tentlike fashion. Or, the operator deliberately may compress the aorta, collapsing its walls between thumb and fingers, in order to palpate the enclosed stiff guide wire.

Once it has been established that the guide wire has passed through the valve, practically all danger of creating a false passage through the aortic wall will have been eliminated. The puncture wound in the ventricle is enlarged slightly, and the head of the closed instrument is inserted boldly into the ventricular chamber. Ventricular bleeding is restrained partly by slight tension on the purse string, applied by the tourniquet, and mainly by an assistant's pressure upon a gauze sponge which is placed loosely to encircle the shaft of the instrument at the site of ventricular insertion.

The instrument head is advanced along the guide wire until it has passed part way through the valve becoming palpable within the aortic lumen, above the valve level to the fingers of the left hand. Even if considerable resistance

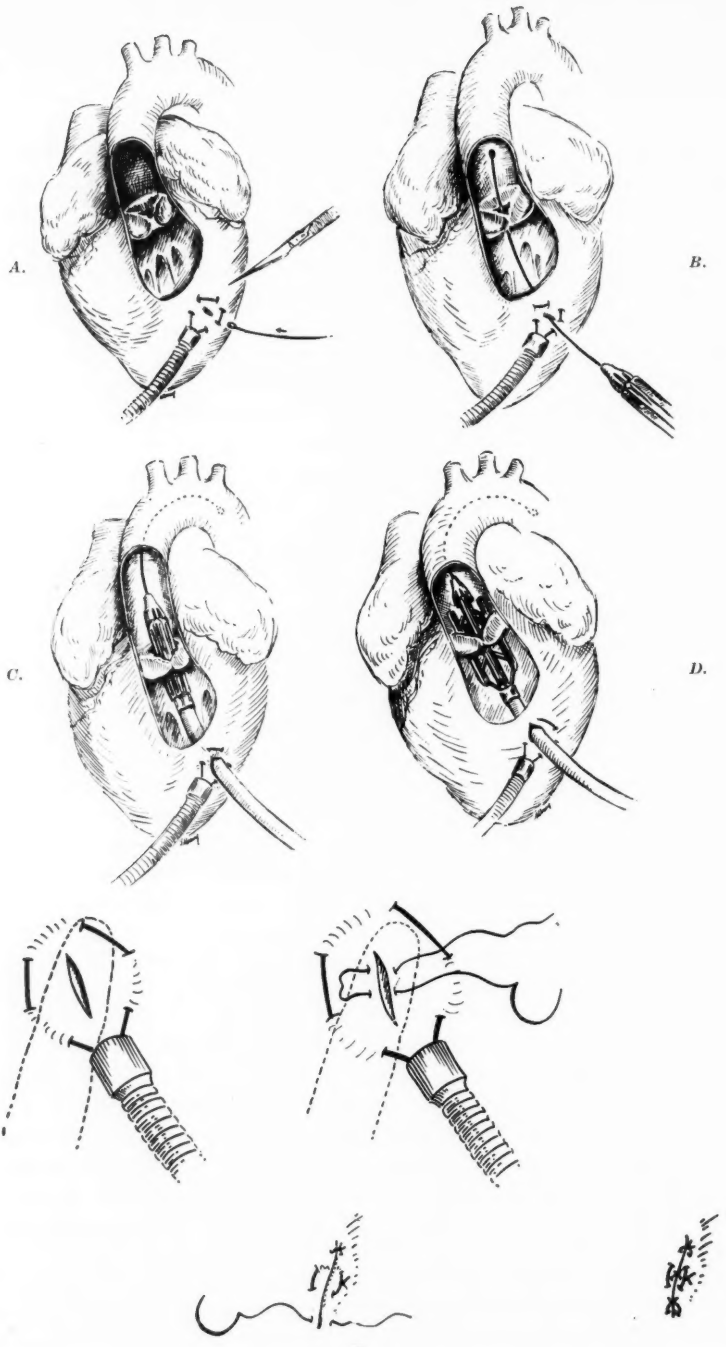


Fig. 18, *A-E.* *(For legend see opposite page.)*

to further advance of the instrument should be encountered at the valve level, the operator need not hesitate to force its entry since the instrument must follow the natural passage along the guide wire. However, it is well first to draw the guide wire back until the olivary tip is palpable only 2 or 3 cm. above the valve. Otherwise there is no certainty that the increased resistance may not be the result of impingement of the olivary tip within a carotid or subclavian arterial orifice. The lock on the handle which prevents the blades from opening is released. Since the head previously will have been adjusted for free rotation upon the central shaft, its triangular cross section presumably will have become aligned automatically with the residual triangularity of the reduced aortic orifice. Hence forceful compression of the handle grips should apply maximal parting or separating force against the central extremity of each line of commissural fusion. The degree of possible expansion of the blades already will have been limited by a setscrew on the handle to the desired range (Fig. 18, *A, B, C, D*).

Because of the possibility of "missing" the valve by inserting the dilator head too far, or not far enough, it is our practice to expand the blades at three different levels. The first is the level that the operator feels is right, the next is placed 2 cm. farther onward (toward the aorta). The last is made at a point thought to be 2 cm. too far back toward the ventricle.

The instrument is closed and is drawn smoothly out of the ventricle and the operator's left index finger is applied lightly over the ventricular puncture. At least one mattress suture and three interrupted sutures are placed deeply to approximate the full thickness of the edges of the ventricular wound. The needles are passed under the finger and the sutures are tied individually (Fig. 18, *E*).

After hemostasis has been secured and vigorous heart action has been established, preparations are made to close the chest. The cut pericardial edges may be closed partially, but a sufficient opening must be left to permit pericardial drainage into the left pleural space. The pleura is drained by a No. 26 F. catheter with extra holes cut into it. It is inserted through a stab wound made in the left seventh or eighth intercostal space at the posterior axillary line. This is connected to a simple water-seal drainage system.

The chest wall is closed using approximating pericostal sutures of heavy chromicized catgut. The muscles and fasciae may be closed with interrupted cotton or continuous chromic catgut. We prefer to close the skin with subcuticular stainless steel wire.

Fig. 18.—Technique of performance of aortic commissurotomy by the transventricular route. *A*, Tiny stab wound made in a selected relatively avascular area (near the left ventricular apex) which has been circumscribed by a purse-string suture. *B*, Extended guide wire is inserted through stab wound into left ventricular lumen and is directed upward along the interventricular septum until it traverses the diminutive aortic opening, the olivary tip becoming palpable within the ascending aorta. C, The ventricular incision is enlarged slightly, and the closed instrument is advanced upon the guide wire until about one-half of the instrumental head is palpable within the aortic lumen (above the valve level). Automatic alignment of the triangular cross-sectional contour of the closed dilator head with the residual triangularity of the valve is assured by the free rotatory mechanism at the junction of the head with the shaft. *D*, Parallel separation of the dilator blades now directs maximal parting force against the central extremities of the fused commissures.

E, Method of digital surface tamponade of the left ventricular wound combined with moderate tension applied to the constricting purse-string suture, and the preferred technique of suturing the myocardium. (From Bailey, Surgery of the Heart, courtesy Lea & Febiger.)

Transaortic (Supravalvular) Digitally Controlled Aortic Commissurotomy.—

Incision: The patient is kept in the supine position after the induction of anesthesia and intubation. The right arm is brought to a 90-degree angle with the trunk at the shoulder. Although both sides of the anterior thorax and both axillae are prepared and draped, the incision is limited to the right hemithorax. In the female, the usual curved inframammary incision is selected and it is extended posteriorly up to the apex of the axilla. In the male, a more transversely directed incision, passing just below the nipple, is acceptable. In either sex, the anterior extremity should not lie farther cephalad than the level of the manubriosternal junction, and here should point nearly in a transverse direction. Then if sternal transection and a left-sided prolongation of the incision should be deemed advisable (perhaps to treat an element of coexisting or created aortic insufficiency), an esthetically curved extension of the wound will be possible.

The fibers of the pectoral muscles for the most part are split approximately in the line of skin incision being divided anteriorly for a limited extent. Bleeding points are picked up meticulously and are controlled either by ligation or by electrocoagulation. The third intercostal space is incised cautiously for entrance into the right pleural cavity. Division of the third costosternal junction affords an appreciable improvement in exposure especially in older individuals.

Great care is taken to dissect the internal mammary artery and vein separately. Both are divided between clamps and the four cut vascular ends are individually ligated with medium silk or cotton. A pair of fixed rib spreaders (Finochietto) is placed with the side bar toward the midline and the intercostal space is widely opened. Separation of the intercostal muscles posteriorly is extended by the blunt pressure of a gauze-covered thumb, facilitated considerably by the disruptive tension provided by the partially opened retractor.

Opening the pericardial sac: The right lung is compressed posteriorly, after dividing any restraining pleural adhesions if necessary. The right phrenic nerve is identified running longitudinally along the pericardium just anterior to the root of the lung. The pericardial sac is incised longitudinally about 1 cm. anterior to and parallel with it up to the level of the junction of the superior vena cava with the atrium, then deviating anteriorly to avoid injuring the former structure. Weighted traction sutures are placed in the posterior cut edge. The opposite extremity of the pericardial incision is directed forward at a right angle, turning at a level which is just caudad to the right atrial appendage. The anterior limit of this section passes under the pericardial fat pad and extends somewhat beyond the right sternal margin. This L-shaped incision results in the mobilization of an upper triangular segment of pericardium, pedicled obliquely forward and upward. Traction sutures placed in its free margins are attached with tension to the crossarm of the fixed rib spreader. This firm anterior pericardial suspension effectively acts

to overcome any tendency of the mediastinal structures and the ascending aorta progressively to migrate toward the left. Such migration would render the exposure to the root of the aorta much less satisfactory.

The ascending portion of the aortic arch now will be visible within the wound, its first 2 or 3 cm. centimeters being overlapped by the right auricular appendage. Direct palpation of the root of the aorta, especially posteriorly, will reveal the typical systolic thrill of aortic stenosis, usually weakening or absence of the normal palpable shock of closure of the valve leaflets, and the diastolic thrill of associated aortic insufficiency if present. Tentative digital compression of the lowest portion of the aorta may reveal underlying masses of valvular calcification if marked. Sometimes a complete ring of calcification about the aortic annulus can be recognized.

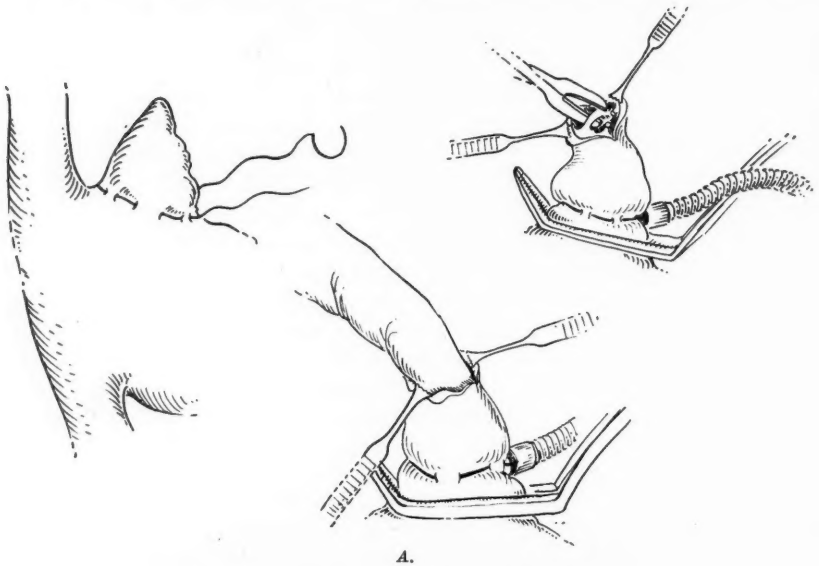
Exploration of the right atrium: It is our practice in these patients first to explore the tricuspid valve by inserting the ungloved left index finger through an incision made in the tip of the right auricular appendage, hemostasis being ensured by a purse-string suture placed about its base. If tricuspid stenosis is found, it is treated immediately in a definitive manner using either the digital splitting or a digitally guided instrumental technique to separate one or more of the fused commissures. It is felt that an orifice sufficiently large to permit two fingers to pass simultaneously (4.5 sq. cm.) should be striven for, provided an opening of this size can be obtained without producing a serious degree of insufficiency. It is felt that the finding of a significant degree of acquired tricuspid stenosis, a lesion apparently never observed in the absence of a coexistent mitral stenotic lesion, renders it mandatory that the mitral valve be explored at this same operative session.

After examining or manipulating the tricuspid valve, the entire inner surface of the atrial chamber and septum is palpated to rule out the possible presence of a septal defect or of anomalous pulmonary venous drainage.

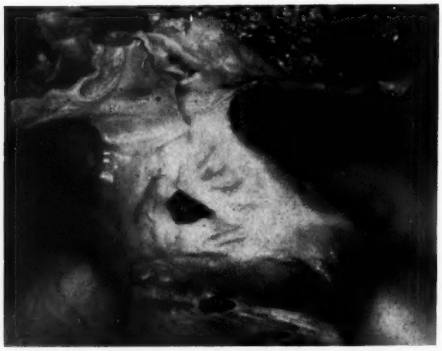
The exact steps of this procedure progress as follows: A nonerushing clamp, usually of the Satinsky type, is closed across the base of the appendage, usually encroaching somewhat upon the true atrial wall. Using a heavy silk or nylon suture with a curved Atraumatic needle, a circumferential purse string is placed about the excluded appendage toward its base. The ends of the suture are incorporated within a Rumel tourniquet. An incision is made in the tip of the excluded appendage, directed in parallel with the blades of the clamp. The lumen of the appendage is flushed with saline and any intervening trabeculae which might interfere with insertion of the finger are divided.

An assistant amputates the portion of the glove which covers the operator's left index finger and the latter is washed with saline and dipped into a dilute solution of heparin (1/1,000). The two lips of the appendage are held up by tissue forceps and the tip of the bare finger is inserted into the opening which first has been filled with saline. As the clamp is released, the finger is advanced into the atrial chamber and the tourniquet is tightened sufficiently to provide hemostasis.

The tricuspid valve is examined and it is determined whether its orifice is competent and of adequate size. If commissural fusion exists, it is evaluated. Then an attempt is made digitally to mobilize the adherent leaflets. If this fails with moderate pressure, one of the guillotine knives used for mitral



A.



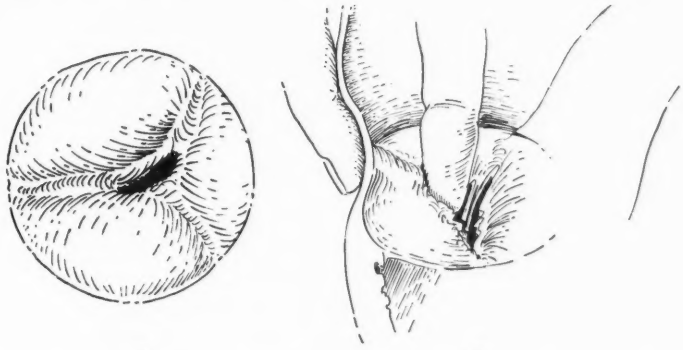
B.

Fig. 19.—*A*, Steps in inserting the operator's ungloved left index finger into the right atrial chamber via the auricular appendage to explore or manipulate the tricuspid valve.

B, Symmetrial commissural obliteration in the tricuspid valve ultimately produces a diminutive central triangular orifice.

commissurotomy is inserted through the appendageal incision along the palmar surface of the finger and one (or more) of the commissures is divided. In general we prefer to avoid anything other than relatively gentle digital manipulation of the anteromedial commissure, and to limit any necessary instrumental separation to the other two (Fig. 19, *A*, *B*, *C*, *D*, *E*).

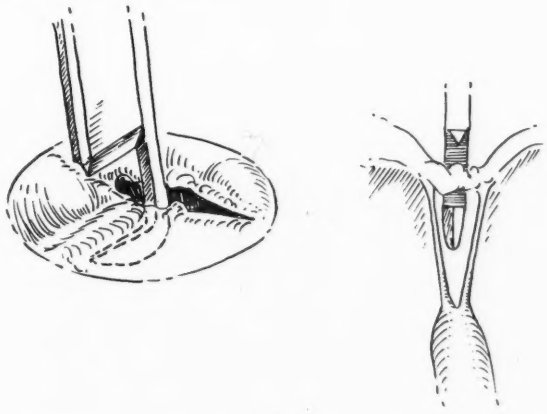
After opening the valve sufficiently, the instrument, if any has been used, is removed from the heart, and the septum and posterior portion of the atrium are explored. The fossa ovalis and the ostium of the coronary sinus routinely are included in this investigation. When the operator is satisfied with the examination, he withdraws his finger, reclamping the appendage.



C. *D.*

Fig. 19.—*C,* Asymmetric fusion of tricuspid valve commissures may lead to an eccentrically located or slitlike residual orifice.

D, An attempt at digital splitting of the fused tricuspid commissures always is indicated before considering an instrumental attack.



E.

Fig. 19.—*E,* Accurate engagement of one of the usual mitral commissurotomy guillotines upon the obliterated valve commissure is essential if created insufficiency is to be avoided. The proper line of section always lies between the two rows of chordae supporting that commissure and is parallel with them. The employment of a bare finger technique—because of its greater tactile sensibility—is essential in ensuring accuracy of placement of the instrument.

The purse string is tightened and tied as the clamp is removed, is passed again around the appendage, and is retied. The distal portion of the appendage is amputated to provide a biopsy specimen, and the cut end is oversewn with a No. 3-0 cotton suture on an Atraumatic needle. This reduces the size and lumen of the right auricular appendage greatly and exposes a significantly greater portion of the root of the aorta for the definitive surgery.

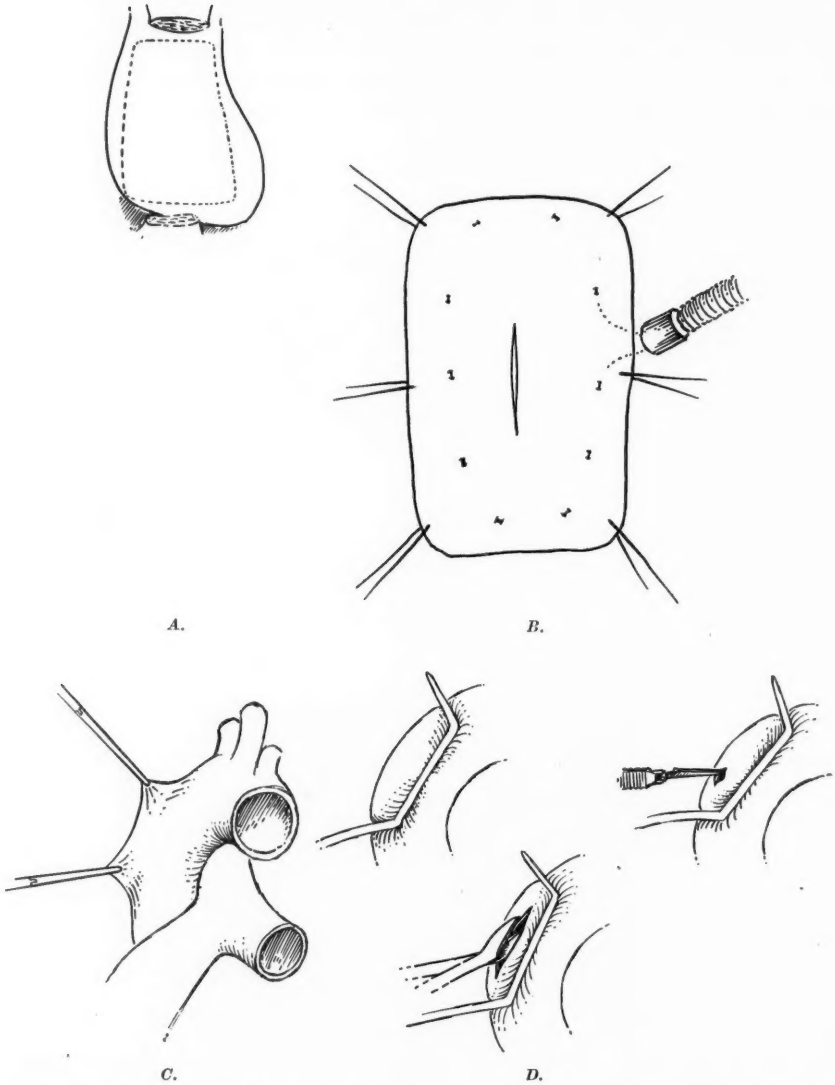


Fig. 20.—*A*, Resection of a large patch of the patient's own pericardium in order to create a pouch to be applied to the ascending aorta. This requires sternal transection and the opening of both pleural cavities. The employment of a preserved pericardial homograft enables the operator to limit the thoracic incision to the right third intercostal space. A patch of plastic fabric (Vinyon or sealed nylon) may be used in a similar fashion. *B*, A 4 cm. incision is made longitudinally in the patch of denuded pericardium or plastic fabric. A purse-string suture of heavy nylon thread is placed about its periphery in such a manner that the line of the suture loops comes to lie farther from the extremities of the incision than from the sides. Only very small loops of suture material are placed upon the smooth (epicardial) internal aspect of the pouch. *C*, The adventitia of the ascending aorta is picked up at selected points with fine hemostats. *D*, A large Satinsky type clamp is applied longitudinally to this portion of the aorta in such a manner as to exclude a sizable portion of its circumference (up to one-half). The excluded portion is incised longitudinally for a distance of 4 cm.

A segment of homograft pericardium obtained, sterilized, and preserved by the method reported by Meeker and Gross[36] for aortic homografts, is thawed out and any excess pericardial fat is dissected away. A rectangular patch 12 by 18 cm. is ideal for this purpose, although a smaller one (above 5 by 8 cm., however) may be used with somewhat more difficulty. While a portion of the patient's own pericardium may be used, this ordinarily necessitates opening both pleural cavities. If the right side of the pericardium is excised for this purpose, it must be replaced by plastic or other type of material to obviate possible postoperative dislocation of the heart into the right pleural space. While we and others, Glenn,[34] Swann[35] have used latex and plastic pouches instead of pericardium, their use often is attended by very serious bleeding from the aortic anastomosis. We feel that the use of a square patch of Vinyon plastic fabric or of nylon fabric in which the central and peripheral cut edges have been flashed in the flame of a match is much to be preferred. The technique is similar to that used for the pericardial patch.

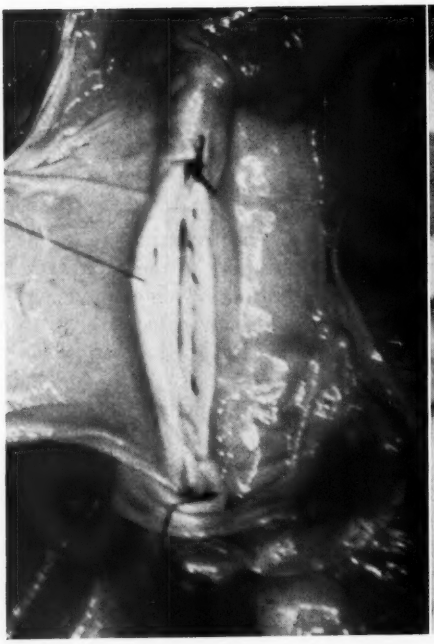
The homograft is spread out upon a moist towel, epicardial surface uppermost. An incision, 4 cm. in length, is made longitudinally in the central portion of the graft. Using a heavy nylon suture with a swedged-on curved needle, the opening in the pericardium is circumscribed at a distance of 2 to 5 cm. from its edges. The bites of the needle are placed in such a manner that very small loops (2 to 3 mm.) of the suture material are placed on the epicardial surface of the membrane, while 1 cm. loops lie upon the roughened pleural surface of this tissue. The extremities of the sutures are caused to emerge from the rough surface. An effort is made to provide a greater margin of distance about the extremities of the opening than at its mid-point. This provision of additional tissue at these points tends to reduce the tension upon the aortic anastomotic sutures at the extremities of the suture line. Several weighted stay sutures are placed about the periphery of the patch to permit it to be held extended.

The adventitia of the right lateral aspect of the ascending aorta is picked up with fine hemostats, and a large Satinsky aortic clamp is placed longitudinally to exclude one-fourth to one-third of the aortic lumen at a site extending upward from a level about 2 cm. above the aorticoventricular junction. A 4 cm. incision is made in the excluded portion of the aortic wall and the lumen is flushed with heparin solution.

Now the pericardial patch is laid over the ascending aorta so that its incision lies upon the aortic opening and the free extremities of the purse string are placed medially. A special mattress suture of No. 4-0 arterial silk is placed at either extremity of the aortic incision in such a way that the angle of junction of the two cut pericardial edges is folded over, and then under the extremity in an imbricating fashion. These sutures are each continued as a running mattress, respectively, along one of the aortic lips, bringing about similar imbrication of pericardium over aortic edge for the full distance of the wound (Fig. 21, *A*, *B*). An extra mattress suture is placed through pericardium and aortic wall just distal to each of the initially placed ones in order to afford additional hemostatic security.

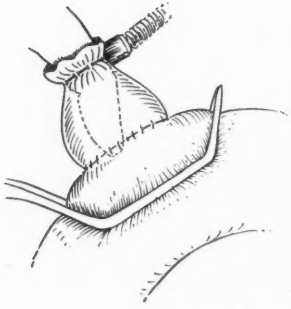
A.

B.



C.

D.



E.

Fig. 21, *A-E*. (*For legend see opposite page.*)

Having completed the attachment of the pericardial patch to the aorta, the purse string is tightened by hand sufficiently to convert it into a "pouch-like" structure arising from the ascending aorta. Then the extremities of the purse-string suture are incorporated within a Rumel tourniquet. The pouch is filled with dilute heparin solution and the tip of the operator's ungloved left index finger is inserted into it. The purse-string suture is tightened about the finger and the aortic clamp is released. The pouch becomes distended with blood and pulsates but only insignificant bleeding should occur either from the line of anastomosis or about the entrance of the finger into the pouch (Fig. 21, *C, D, E*). If such bleeding should be marked, the aorta must be reclamped and the leak repaired before going ahead with the definitive surgery.

The operator advances his left index finger into the aorta as the tourniquet is loosened slightly. Soon the finger tip approaches the valve and detects the jet of blood emerging from the ventricle, with each systole. By following up the systolic jet, the finger tip will soon reach the diminutive valve orifice and be able to palpate it momentarily. This may be repeated from time to time being careful never to block it for more than 3 heartbeats at a time, and allowing a somewhat longer interval of free circulation between such examinations. Sometimes the valve opening may be so small that it can be recognized only with the greatest difficulty.

An attempt is made to recognize the various commissures, and the concave upper surfaces of the cusps, and the sinuses of Valsalva are explored. Having estimated the likelihood of restoration of some valvular function the finger tip is inserted into the diminutive opening, splitting it somewhat if necessary, and direct digital pressure is made first against that commissure which is considered most favorable for separation. Within less than 3 heartbeats the pressure is released and the finger is drawn out of the valve orifice into the aorta for a few moments. If splitting of this commissure has taken place and been adequate, attention is turned toward another one. If separation has not begun, or has been incomplete, another attempt is made to split it digitally, this time with the aid of external counter pressure, applied by the gauze-covered fingers of the right hand. Sometimes hooking the intra-aortic finger tip beneath the valve commissure and applying upward traction will prove a more effective way of breaking the cross fusion of the cusps. However, this is a somewhat dangerous maneuver and may lead actually to partial detachment of one of the leaflets from the annulus fibrosus with resultant ensuing regurgitation (Fig. 22, *A, B*).

It is felt that if one commissure has been split to the aortic wall, and another one-half way, a minimally adequate valve passageway will have been established. Nevertheless, the functional result may not be satisfactory if the

Fig. 21.—*A*, Method of folding cut edge of central incision in pericardial or plastic fabric patch over lip of aortic incision. Continuous through-and-through mattress sutures produce a permanently imbricated or mortised juncture of the tissues. Such anastomoses do not leak even under the strain of full aortic pressure. *B*, Actual photograph showing implementation of this concept. *C*, By drawing upon the purse string, a pouch without a lateral seam is formed. *D*, Insertion of ungloved left index finger of operator into pouch after filling it with saline solution. Purse-string tension is increased to prevent blood loss when aortic clamp is released. *E*, Actual photograph showing bare finger tip in pouch just before removing aortic clamp. (From Bailey, Surgery of the Heart, courtesy Lea & Febiger.)

valve components are relatively stiff and nonmobile. However, the mere fact of calcification need not necessarily indicate an incapacity of the valve to function. Provided there is significant flexibility in the basal portion of the valve or in the aortic wall itself at the site of valvular attachment, a considerable amount of normal type cusp movement may be established. In all cases as much anatomic valvular mobilization should be accomplished as is readily possible.

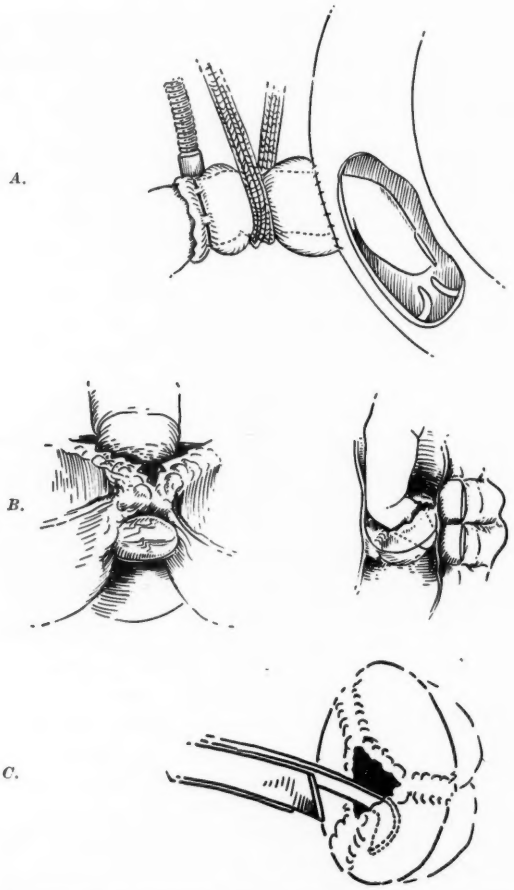


Fig. 22.—*A*, Finger tip is advanced into the aortic lumen and finally to the aortic valve level to explore its pathologic condition. A second line of hemostasis may be established by tensing a strip of gauze placed around the pouch proximal to the purse-string suture. *B*, In nearly half of the patients, one or more of the fused commissures can be separated by digital pressure, perhaps aided by external counterpressure with the opposite (right) hand. *C*, One of the mitral commissure guillotine knives may be inserted along the finger and through the pouch to divide a resistant commissure. Extreme accuracy of application is essential. (From Bailey, Surgery of the Heart, courtesy Lea & Febiger.)

Should the digitally established valvular enlargement be deemed to be inadequate or negligible, instrumental help becomes mandatory. In some cases one of the usual mitral commissurotomy guillotines may be inserted through

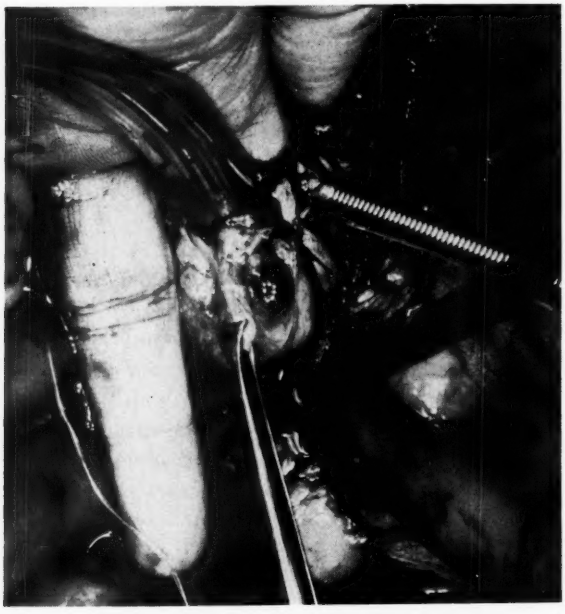
the appendage along the finger. This technique was first applied by Swann.[35] It is passed through the valve orifice, is applied against an appropriate commissure, and is opened. The hooked underblade is engaged accurately under the line of valvular fusion, and then the instrument is closed by advancing the sliding guillotine-type blade. If accurately performed, a good anatomic separation of the valve elements may be achieved. However, the least inaccuracy of application may create a severe degree of aortic insufficiency (Fig. 22, *C*).

In many cases one may prefer to insert a two or three bladed retrograde aortic dilator through the opening in the pouch along the palmar surface of the operator's index finger. This may be accomplished without encountering excessive hemorrhage by first applying a constricting loop of unrolled gauze about the basal portion of the pouch. This permits relaxation of the distal purse string so that the lead end of the closed dilator may be placed within the opened upper portion of the pouch. The purse string is tightened about the instrument and finger, and the gauze is loosened slightly (but not completely, as it aids in the hemostasis). The instrument is advanced cautiously until its olivary tip lies just above the aortic valve. The intra-aortic finger locates the valve orifice (by the emerging systolic jet) and guides the tip of the closed instrument through it.

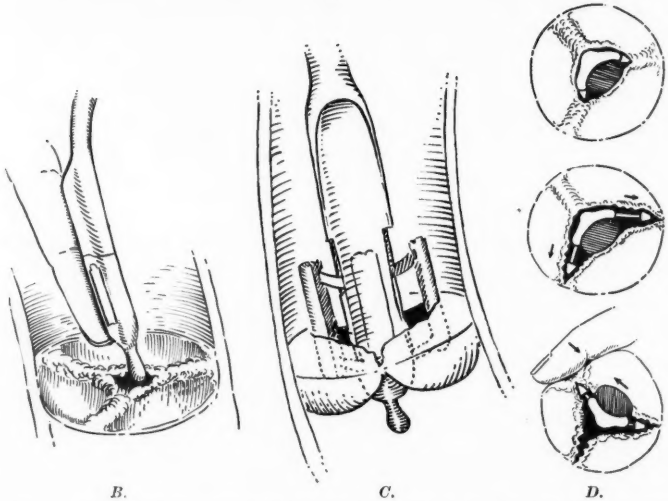
This technique may prove unusually difficult in some of the "clamshell" valves where there is an almost unrecognizably narrow slit which can be detected only by palpating the emerging systolic jetlike stream of blood. Here, the finger tip is poised on the valve edge in the region of the maximal jet. By timing with the cardiac cycle the flat edge of the finger tip may be inserted (perhaps forcibly) into the maximally open valve slit at the exact moment of beginning systole (beginning onset of the jet). To date this maneuver has been possible (and successful) in every patient operated upon by the supravalvular approach.

Once the finger tip has been inserted into a very small valve orifice (and has dilated it somewhat) it only requires exact timing to replace it by the tip of the closed instrument as the finger tip is withdrawn. Deeper insertion to apply the shoulder and dilating portion of the instrument into the valve may entail the use of considerable force.

The finger and dilator are turned in such a way as to line up one of the parting blades against the commissure deemed most favorable for blunt disruption. The other blade automatically becomes lined up with another commissure. By cautious compression of the hand grips of the instrument, the two blades apply pressure against the appropriate commissures, nearly always splitting at least one of them. Any intimation that one blade has begun to make pressure directly against the aortic wall or that valvular separation has begun at a point other than at a commissure is an indication for re-evaluation of the problem or termination of the procedure. By rotating the instrument 120 degrees, the third commissure may be attacked. After use of the instrument further digital splitting may become possible, just as it often is in the



A.



B. *C.* *D.*

Fig. 23.—*A*, Two-bladed retrograde dilator, illustrated in Fig. 17, is inserted through the pouch along the palmar surface of intra-aortic finger to separate resistant valve commissures. *B*, Olivary tip of dilator is guided by intra-arterial finger to the diminutive valve orifice by the technique of following up the pulsatile jet of blood emerging from the nozzle-like valve opening. *C*, After the dilator mechanism has been forced into the valve orifice and has become aligned with two of the commissures, the blades are caused to expand by compressing the handles of the dilator, thus applying splitting pressure simultaneously against two of the fused valve commissures. *D*, Representation of method of applying parting pressure against each of the three valve commissures. (From Bailey, Surgery of the Heart, courtesy Lea & Febiger.)

course of a digitally guided mitral commissurotomy. Indeed the whole procedure closely resembles that of the now well-established comparable surgery of the stenotic mitral valve (Fig. 23, *A, B, C, D*).

Upon the termination of valvular instrumentation, the operator reassures himself (by intra-arterial examination) that the blades of the dilator are completely retracted (closed) and then simply withdraws the instrument from the vessel and pouch. Any attempt at withdrawal with the blades even slightly expanded is fraught with great danger of engagement upon a portion of the valve or of laceration of the aortic wall itself.

The finger tip is withdrawn into the pericardial pouch and a generous portion of the aortic wall, beyond the basal attachment, is clamped with the special Satinsky clamp. The attaching sutures at the base of the pouch are cut away and the aortic incision is repaired using one row of continuous mattress sutures of No. 4-0 arterial silk, and one more superficially placed row of continuous over-and-over sutures. After releasing the clamp, simple digital pressure with a gauze pad will suffice to control any oozing from the stitch holes.

Exploration of the mitral valve: Providing the patient's condition is satisfactory after the completion of the closure of the aortic wall, it is felt that a digital exploration of the mitral valve is indicated routinely. All too often in spite of the exhibition of all the usual clinical and physiologic methods of investigation, a dynamically significant mitral valvular lesion may be overlooked in a patient with preponderant aortic disease. Should the operator fail simultaneously to correct such a lesion, especially if it is a stenotic one, significant over-all clinical improvement may not occur, since the cardiac output will not increase appreciably as a result of relief of the aortic obstruction alone. Hence, since mitral stenosis is present in about one-half of all patients operated for aortic stenosis, we feel that such a coexistent lesion should be ruled out in all cases, if the patient's condition permits, by direct digital examination of the mitral valve. Naturally, if a strong suspicion of coexisting mitral stenosis has been expressed by some of the medical attendants, one's desire to rule it out definitively is enhanced. The presence of atrial fibrillation usually indicates associated mitral disease since it rarely appears in uncomplicated aortic valvular disease but is very common with mitral lesions. The previous finding of a serious degree of tricuspid stenosis is considered to be a pathognomonic indication of the presence of mitral stenosis.

The interatrial groove is exposed just to the right of, and posterior to, the superior vena cava. It is dissected apart for a distance of about 1.5 cm. Then a small bite purse-string suture is placed widely (5 to 6 mm. distance) about this area of dissection, using a curved needle swedged on a heavy nylon suture. Care is taken that each pass of the needle penetrates into one of the atrial lumina. The ends of the suture are incorporated within a Rumel tourniquet for reliable flexible hemostatic control (Fig. 24, *A, B, C, D*). A No. 3-0 cotton suture is placed and tied at each extremity of the dissection to facilitate subsequent reparative suturing.

The interatrial septum is dissected somewhat farther, perhaps to a depth of 2 cm. taking great care not to break, inadvertently, into the right atrium. The ungloved tip of the operator's left index finger is inserted into this dissected area. A large knife blade (Bard-Parker No. 20) on a detachable handle is laid upon the palmar surface of this bare finger and is advanced over the tip to penetrate through the left or lower half of the septum and to enter the chamber of the left atrium. The knife is withdrawn with care to avoid cutting the purse-string suture which is kept loosened at this time. The finger tip seeks out the site of hemiseptal puncture, tamponading it and then advancing through it to the region of the mitral valve. This valve lies farther from the right-sided site of cardiac entrance than it does when approached through the more usual left-sided incision, and, therefore, may necessitate the insertion of nearly the whole length of the finger into the atrium (Fig. 25, *A, B, C, D*).

The valve slit runs approximately horizontally with the patient in the supine position, and, hence, nearly in line with the finger. This is at right angles to its general line of direction when examined from the left lateral approach. After exploring and evaluating the anatomic and functional condition of the valve, thought is given to the technique of performing a commissurotomy if mitral stenosis has been found or some type of repair if the valve is incompetent. The latter is a more major undertaking and will not be detailed in this paper. However, the former is so readily and logically accomplished from this approach that it will be given at this time.

Mitral commissurotomy from the right side: The tip of the bare index finger is directed first against the least accessible (anterolateral) commissure of the valve. In a fair percentage of cases (20 to 30 per cent) anatomic splitting will take place even without the exhibition of much pressure. However, really strong disruptive pressure against this commissure can scarcely be applied from this approach since effective external digital counterpressure is well nigh impossible. Hence, the use of an appropriate instrument may become necessary, unless the posterior commissure can be opened sufficiently by itself to provide an adequate final passageway (1½ to 2 fingers or 3.0 to 4.5 sq. cm. cross-sectional area).

Digital separation of the very readily accessible posterior commissure is accomplished much more easily from this approach than from the more usual left lateral one through the left auricular appendage. As in the digital performance of commissurotomy, by any approach, it must be done only a little at a time by intermittent application of pressure, in order to avoid a sudden great extension of the separation which might be complicated by severe regurgitation. Probably the posterior commissure can be split digitally to some degree in at least 50 per cent of patients thus operated upon.

When an incompletely satisfactory opening is obtained by these maneuvers, instrumental aid becomes mandatory. Our preferred instruments are essentially longer versions of the usual commissurotomy guillotines* modified as suggested by Nichols[43] so that they may be used also to divide a directly adherent papillary muscle should it prove necessary.

*Obtainable from George P. Pilling & Son Co., Philadelphia, Pa.

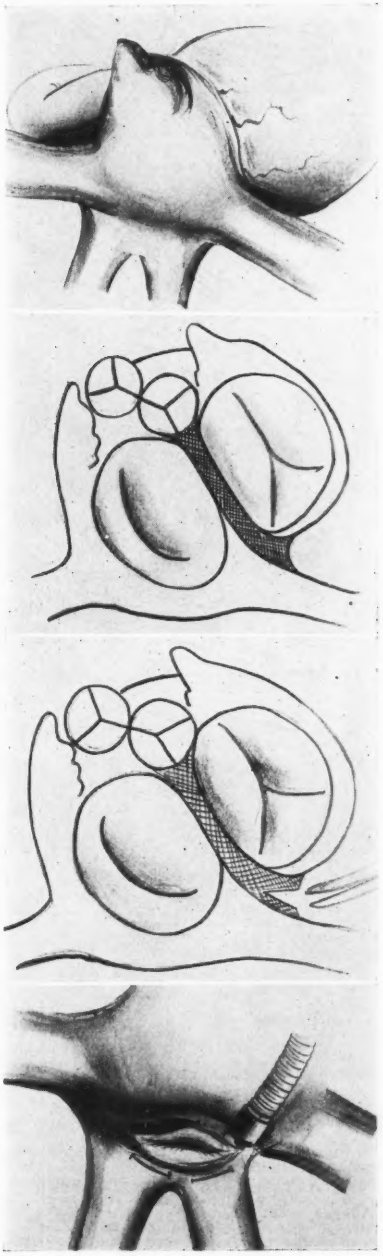
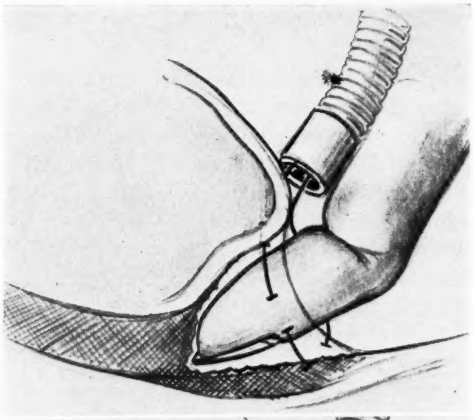
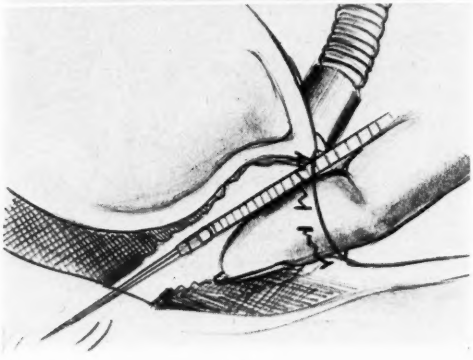


Fig. 24.—*A*, Pertinent anatomy concerned with the performance of mitral commissurotomy from the right side, via the interatrial groove. *B*, Cross section of the heart made just above the level of the four valves. The heavily shaded diagonal line represents the interatrial septum which is to be separated or split into two halves. *C*, Beginning dissection of the interatrial groove and septum. *D*, Not over 1.5 cm. of the length of the interatrial groove is separated. A purse-string suture of heavy nylon thread is placed about this area of dissection, taking care that each bite penetrates into the respective atrial lumen. A Rumel tourniquet is employed to provide flexible hemostatic tension.

A.





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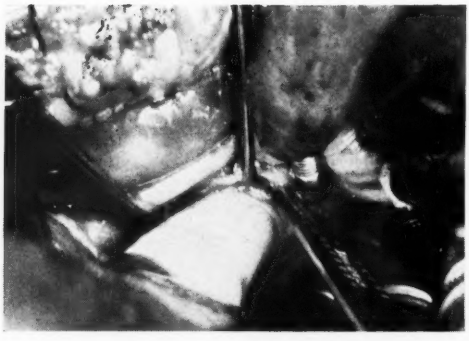

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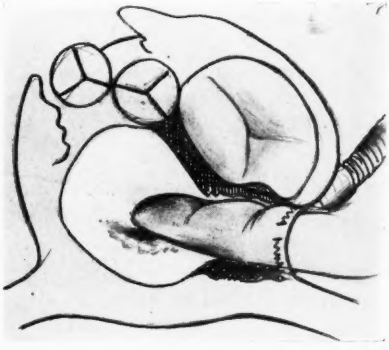


D.

Fig. 25.—*A,* The dissection of the interatrial septum is carried to a depth of 1.5 cm., in part by digital separation.

B, A large-bladed knife (Bard-Parker No. 20) is laid upon the palmar surface of the bare finger tip as it lies in the furrow of dissection. The direction of insertion of the knife is angulated and it is advanced over the tip of the finger until it pierces the left side of the separated septum. It should not penetrate farther than 1 cm. within the left atrial chamber lest it pierce the posterior (opposite) atrial wall.

C, Actual photograph of knife puncturing into left atrial chamber via the interatrial groove. Care must be taken to avoid dividing the encircling purse-string suture which is kept loose during this maneuver. A gush of bright red blood indicates entry into the left atrium.

D, As knife is withdrawn, the operator's finger tip is advanced, seeking and finding the site of puncture of the left half of the atrial septum and penetrating through it. The septal opening will be found to be somewhat too small for the finger, thus providing a secondary hemostatic line of constriction.

For the anterior commissure the left-sided guillotine is inserted into the heart along the bare palmar surface of the intracardiac finger as the anterior lip of the dissected sulcus is held upward with hand-held tissue forceps. It is passed through the valve orifice in the closed position and is applied against the anterior (less accessible) commissure taking care that it comes to lie between the two rows of chordae, respectively, supporting the two leaflets. These lie more posteriorly (toward the vertebrae) than would be expected. The sliding blade is retracted by the right hand while the intracardiac finger tip presses the edge of the instrument against the extremity of the valve orifice. As the sliding blade is retracted the hooked underblade slips beneath the commissure. At this point the handle of the instrument is maneuvered so that the cutting blades are directed horizontally. Pressure of the right thumb closes the instrument, the sliding blade shearing through 4 mm. of the thickened commissural tissue. If the tissue is deemed thick and fibrous enough to justify a

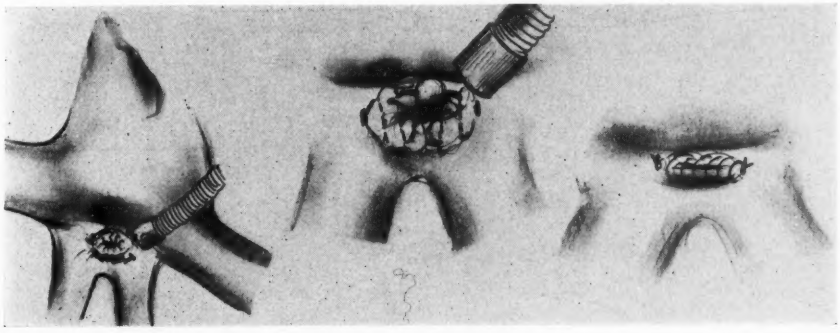


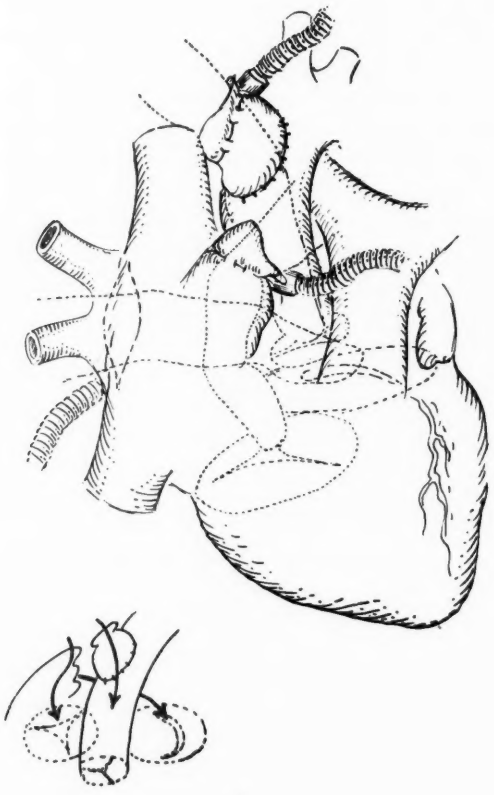
Fig. 25.—*E*, Steps in the closure of the dissected portion of the interatrial septum after the finger is withdrawn. Purse-string tension provides initial hemostasis. Application of a row of continuous mattress sutures approximates the dissected lips of the interatrial groove. Final oversewing stitches reinforce closure. No attempt is made directly to suture the edges of the incised left half of the septum. Purse string may produce puckering of inner atrial wall and should be removed.

second cut the sliding blade is drawn back without removing the instrument from the valve, permitting the hooked underblade to pass further under the commissural tissue. Maintenance of finger tip pressure against the cutting extremity of the instrument ensures exact placement of the instrument into the apex of the previous incision.

Often the operator will prefer to remove the instrument from the valve momentarily, while he applies tentative disruptive pressure to the partially incised commissure. If separation then begins with digital pressure, the instrument is removed from the heart and the commissurotomy is completed by a splitting technique.

If an inadequate valve opening has resulted from these efforts, instrumentation of the posterior commissure is in order. This is not attended by the same considerable risk of production of insufficiency which has been experienced by nearly all operators in attempts at posterior commissural instrumentation from the left side. The right-sided guillotine is inserted into

the left atrium in the manner used previously for the left-sided one. It is passed into the valve orifice in a closed position and is opened while its edge is being pressed back against the posterior commissure. Again great care must be taken to apply it so that the hooked underblade becomes placed accurately between the two rows of supporting chordae. This commissure is cut in the same manner as is the anterior, and the commissurotomy is continued digitally if and when this method of leaflet mobilization becomes effective.



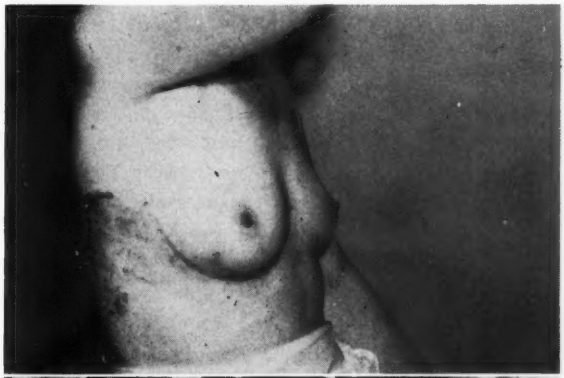
A.

Fig. 26, *A*.—Sketch showing how all three of the valves which are subject to clinical rheumatic involvement may be approached from a right-sided thoracic incision for exploration and corrective surgery. The aortic valve may be approached through a pouch sutured on the ascending aorta, the mitral through the dissected interatrial groove, and the tricuspid through the incised right auricular appendage.

Subvalvular cross fusion of the chordae or of the papillary muscles themselves should be handled in exactly the same manner as they are through the left-sided approach to this valve. Nichols[44] has described these methods in some detail. Since the guillotines designed especially for the right-sided approach have been made to include the principle of his Papillotome, no additional instrument of that type is necessary.

In general, when instrumentally dividing a commissure from the right, one must remember that, in this position, the extremities of the deformed commissures are not usually directed above the horizontal. Hence, the line of the cutting blades of the instrument, when dividing either commissure,

B.



C.

Fig. 26, *B* and *C*.— *B*, Photograph of right-sided incisional scar in patient subjected to simultaneous corrective surgery of all three valves. *C*, Photograph of the less esthetic posterolateral thoracic incision employed in the usual left-sided approach to the mitral valve.

ordinarily should be directed horizontally rather than upward into the substance of the preponderantly important septal leaflet. However, the line of valve fusion then turns upward at either extremity so that a second cut should be deviated in that direction.

When the definitive mitral surgery has been completed, the finger is withdrawn gradually from the heart, the purse-string suture being tightened progressively. When the finger has been removed entirely, the puckered anus-like opening between the atria is sutured together with cotton (3-0), using one row of continuous mattress sutures and one row of simple running sutures. No attempt is made to suture the slitlike opening which has been created in the left side of the interatrial septum (Fig. 25, *A, B, C, D, E,* and Fig. 26, *A*). The tourniquet and the purse string are removed.

The pericardium is flushed with saline and is repaired loosely with interrupted cotton sutures. The pleura is flushed and is drained with a No. 26 F. urethral catheter with additional holes cut into it. This is passed through a stab wound in the right eighth intercostal space, posterior axillary line, and is connected with a water-seal drainage system.

The ribs are approximated with heavy pericostal sutures of chromicized catgut. The muscles and fasciae of the chest wall are closed in a similar manner. The skin is closed with continuous subcuticular stainless steel wire. This incision produces a much shorter and cosmetically more acceptable scar than does the older left posterolateral one (Fig. 26, *B, C*).

RESULTS OF AORTIC COMMISSUROTOMY

Naturally, it is impossible to present the desirable long term follow-up in a series of patients subjected to aortic commissurotomy since the first of them was operated upon (by a reasonably acceptable technique) only three years ago (April 4, 1952). Furthermore, the actual clinical and statistical results obtained in a beginning series in a pioneering effort directed toward the alleviation of a previously inoperable lesion should be evaluated both generously and realistically. The nearly continuous changes in methods of case selection, in surgical technique, and in medical management have vitiated many possibilities of nice and meticulous comparison. The coexistence of other valvular conditions in many of the patients has added other variables to our consideration. In retrospect, we can see that there have been errors in concept, in judgment, and in technique. The knowledge which now permits their recognition and correction, however, could scarcely have been obtained other than by the frequently painful one of trial and error.

A total of 287 patients with aortic stenosis have been operated upon since March 6, 1950. All but those with essentially "pure" aortic stenosis and combined aortic and mitral stenosis operated upon by the triradiate transventricular dilator or through the supravalvular approach have been eliminated from further critical evaluation. Twenty-one and three-tenths per cent of the patients were operated upon by the much preferred supravalvular or transaortic approach using an artificial "aortic appendage" and a digital or a digitally controlled technique. The operative mortality and the postoperative results obtained with the transventricular operations appear on Tables I, II, IV, and V.

TABLE IV. RESULTS OF 68 PATIENTS OPERATED UPON FOR AORTIC STENOSIS BY THE TRANSVENTRICULAR APPROACH. ONE- TO THREE-YEAR FOLLOW-UP

AMERICAN HEART ASSOC. CLASS	NUMBER OF CASES	OPERATIVE DEATHS	LATE DEATHS	SURVIVORS					
				MARKEDLY IMPROVED	IMPROVED	SAME	WORSE	PERCENTAGE IMPROVED OR MARKEDLY IMPROVED (%)	LOST TO FOLLOW-UP
I	3	1 (33.3%)	-	-	2 (100%)	-	-	100	
II	19	2 (10.5%)	2 (10.5%)	3 (23%)	8 (61.5%)	1 (7.7%)	1 (7.7%)	84.5	2
III	44	16 (36.4%)	8 (18.3%)	4 (21%)	12 (63.2%)	1 (5.3%)	2 (10.5%)	84.3	1
IV	2	0	2 (100%)	-	-	-	-		
Total	68	19 (28%)	12 (17.7%)	7 (20.6%)	22 (64.7%)	2 (5.9%)	3 (8.8%)	85.3	3

TABLE V. RESULTS OF 87 PATIENTS OPERATED UPON FOR MITRAL AND AORTIC STENOSIS BY THE LEFT-SIDED APPROACH. SIX-MONTH TO THREE-YEAR FOLLOW-UP

AMERICAN HEART ASSOC. CLASS	NUMBER OF CASES	OPERATIVE DEATHS	LATE DEATHS	SURVIVORS					
				MARKEDLY IMPROVED	IMPROVED	SAME	WORSE	PERCENTAGE IMPROVED OR MARKEDLY IMPROVED (%)	LOST TO FOLLOW-UP
I	0	-	-	-	-	-	-	-	-
II	14	-	1 (7.2%)	2 (15.4%)	7 (53.8%)	2 (15.4%)	2 (15.4%)	69	-
III	66	14 (21.2%)	3 (4.5%)	19 (39.6%)	25 (52.1%)	1 (2.1%)	3 (6.2%)	92	1
IV	7	2 (27.6%)	-	3 (60%)	2 (40%)	-	-	100	-
Total	87	16 (18.4%)	4 (4.6%)	24 (36.4%)	34 (51.5%)	3 (4.5%)	5 (7.6%)	88	1

TABLE VI. RESULTS OF SURGERY UPON 47 PATIENTS OPERATED UPON FOR AORTIC STENOSIS BY THE TRANSAORTIC APPROACH

AMERICAN HEART ASSOC. CLASS	NUMBER OF CASES	OPERATIVE DEATHS	LATE DEATHS	SURVIVORS				
				MARKEDLY IMPROVED	IMPROVED	SAME	WORSE	PERCENTAGE IMPROVED OR MARKEDLY IMPROVED (%)
I	2	0	0			2 (100%)		
II	12	1 (8.3%)	3 (25%)	1 (12.5%)	5 (62.5%)	0	2 (25%)	75
III	30	6 (20%)	5 (16.6%)	3 (15.8%)	14 (73.6%)	1 (5.3%)	1 (5.3%)	89.4
IV	3	0	0	1 (33.3%)	2 (66.6%)	-	-	100
Total	47	7 (14.7%)	8 (17%)	5 (15.6%)	21 (65.6%)	3 (9.4%)	3 (9.4%)	81.2

TABLE VII. CAUSES OF DEATH IN PATIENTS OPERATED UPON FOR PURE AORTIC STENOSIS BY THE TRANSAORTIC TECHNIQUE

Cardiac arrest (3 with severe hemorrhage)	4
Ventricular fibrillation	1
Uremia	1
Creation of severe insufficiency	1
Total	7

Even though 3 of these deaths are believed to be due largely to inexperience of the operator with this new technique, both the early and later operative mortality has been distinctly lower than that associated with the transventricular approach. Furthermore, a detectable increase in a preoperatively recognized element of aortic insufficiency was noted in only 1 patient in this series. Follow-up has been possible in all of those surviving surgery but only in 57 per cent has this follow-up been longer than six months.

The operative mortality experienced in 47 consecutive operations by the transaortic approach for aortic stenosis alone is expressed in Table VI and VII. That in 14 consecutive combined operations using this approach and a right-sided mitral technique is shown in Table VIII.

TABLE VIII. OPERATIVE MORTALITY IN TRANSAORTIC APPROACH AND RIGHT-SIDED MITRAL COMMISSUROTOMIES IN COMBINED MITRAL AND AORTIC STENOSIS

AMERICAN HEART ASSOCIATION CLASS	NUMBER OF CASES	HOSPITAL DEATHS	OPERATIVE MORTALITY RATE
I	0	0	0
II	5	0	0
III	9	1	11.1%
IV	0	0	0
Total	14	1	7.1%

Alterations achieved by surgery with respect to various objective findings and subjective symptoms are detailed in Tables IX and X.

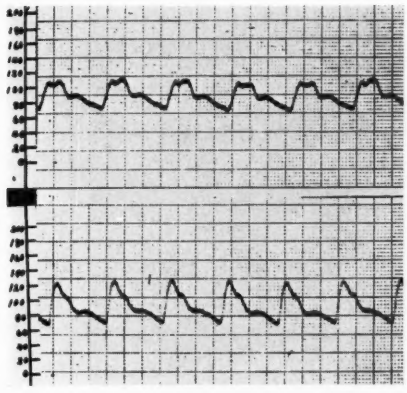
TABLE IX. SIX-MONTH FOLLOW-UP EVALUATION OF PRE- AND POSTOPERATIVE SYMPTOMS OF PATIENTS OPERATED UPON FOR "PURE" AORTIC STENOSIS (SUPRAVALVULAR APPROACH)

	DYSPNEA	SYNCOPE	FATIGUE	PAROXYSMAL NOCTURNAL DYSPNEA	ORTHOPNEA	ANGINA	PERIPHERAL EDEMA	VERTIGO
Present Preoperatively	90%	47%	81%	33%	28%	43%	24%	43%
Postoperatively	Improved or Absent	Present	Present	Present	Present	Present	Present	Present
	62%	10.5%	33%	9.5%	14%	4.8%	9.5%	14%

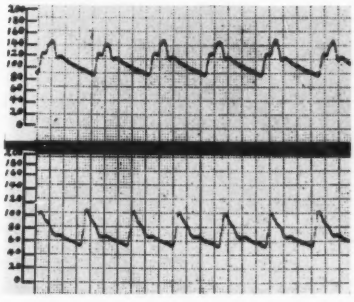
TABLE X. OBJECTIVE POSTOPERATIVE CHANGES FOLLOWING AORTIC COMMISSUROTOMY (AT LEAST BY SIX MONTHS)

CHANGES POSTOPERATIVELY	AORTIC SYSTOLIC MURMUR (%)	AORTIC DIASTOLIC MURMUR (%)	AORTIC SECOND SOUND (%)	HEART SIZE X-RAY (%)	PULSE PRESSURE (%)
Absent	–	18.2	–	–	–
Diminished	54.5	4.5	–	13.6	5.3
Unchanged	41	45.5	31.8	72.8	52.6
Increased	4.5	31.8	68.2	13.6	42.1

Alterations imparted to the brachial arterial pressure curves are shown in Fig. 27, *A*, *B*, *C*.



A.



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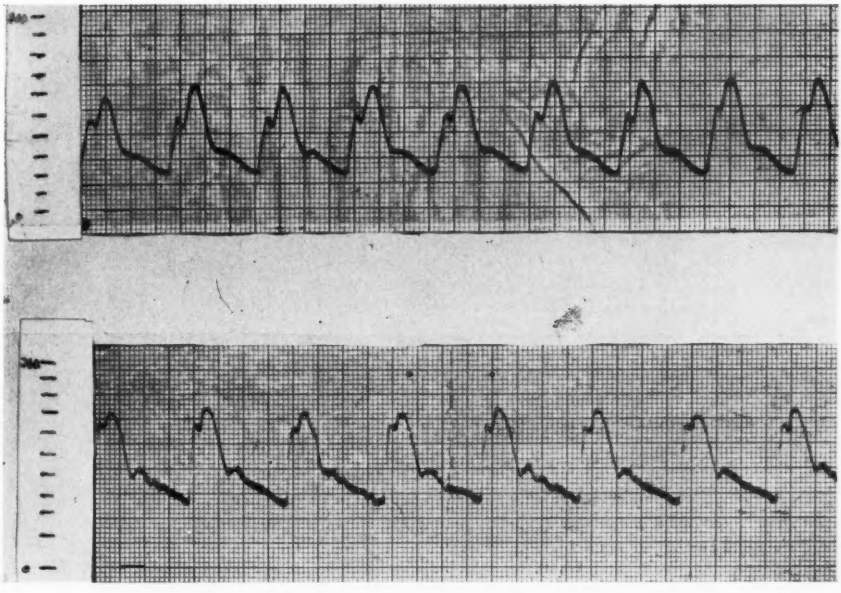
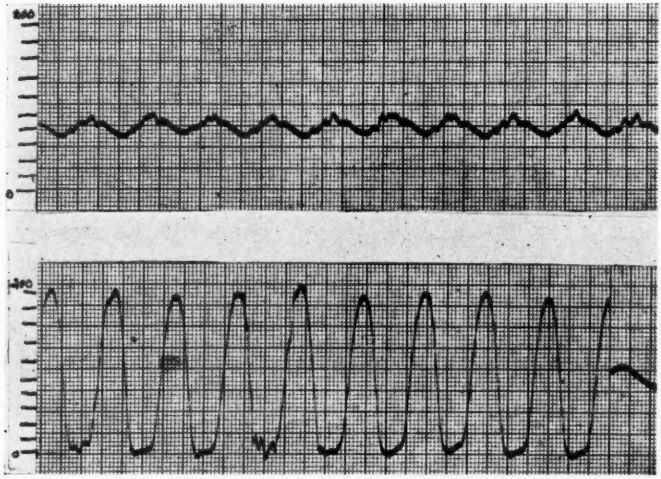
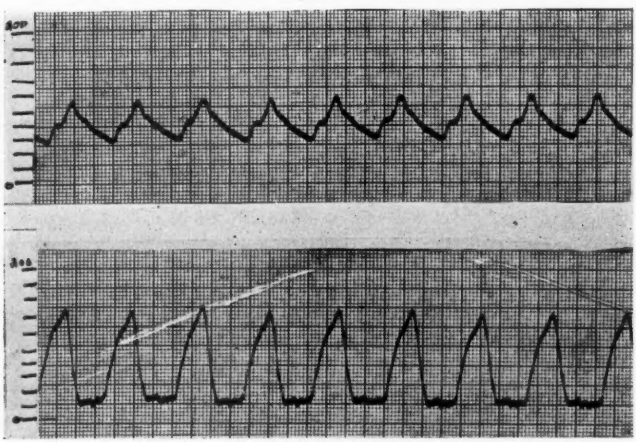


C.

Fig. 27.—*A*, Abolition of the anacrotic notch (double-summit curve) in brachial arterial pressure tracing by satisfactory aortic commissurotomy. Upper curve, preoperative; lower curve, postoperative. *B*, Abolition of the anacrotic notch on the ascending limb of the brachial arterial tracing by satisfactory aortic commissurotomy. Upper curve, preoperative; lower curve, postoperative. *C*, Rise of the anacrotic notch on the ascending limb of brachial arterial tracing from a lower to a higher level, following aortic commissurotomy. This is considered as evidence of improvement (lessening) in the aortic stenosis although it is evident that a significant amount persists. (From Bailey, Surgery of the Heart, courtesy Lea & Febiger.)



A.

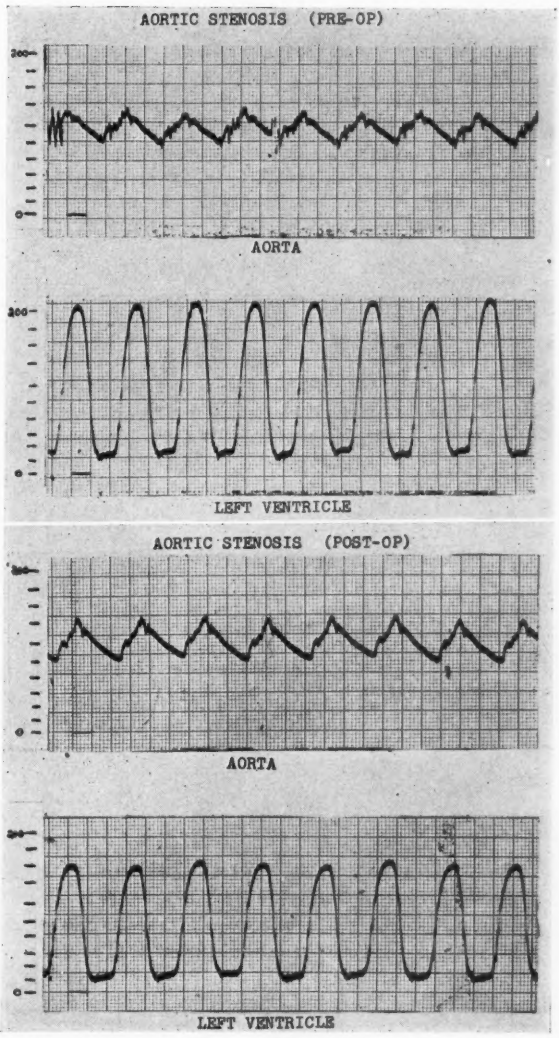


B.

Fig. 28.—Precommissurotomy. *A*, Preoperative differential between aortic and ventricular pressures in W. E. (obtained by direct puncture at surgery).

B, Comparative postoperative pressures in W. E. showing abolition of gradient, obtained immediately after supravalvular digital aorbic commissurotomy.

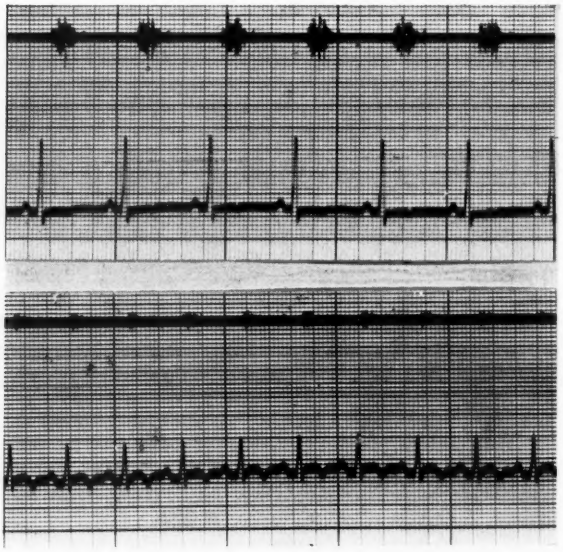
C.



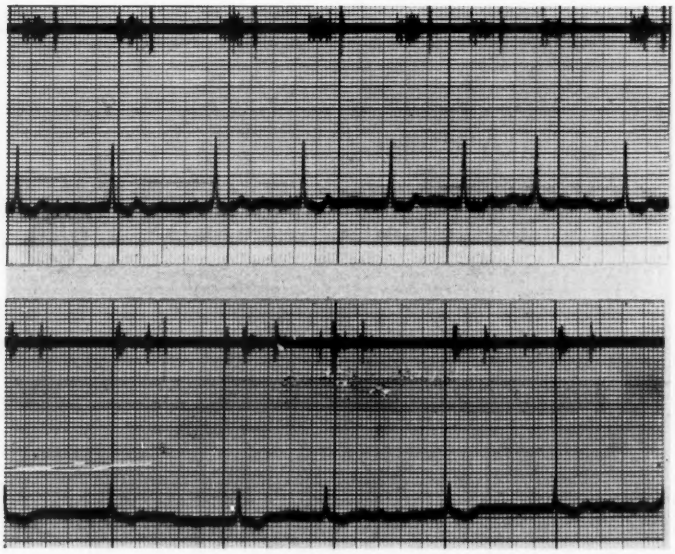
D.

Fig. 28, *C* and *D*.—*C*, Pressure tracing obtained from the aorta and left ventricle in N. F., prior to aortic commissurotomy.

D, Comparative postoperative tracings showing reduction in systolic gradient but failure to achieve complete abolition by performance of supravalvular instrumental commissurotomy.



A.



B.

Fig. 29.—*A*, Phonocardiograms in A. W., showing a great postoperative reduction in the intensity of the systolic murmur of aortic stenosis. There is no evidence of restoration of the aortic second sound. (Upper part, preoperative; lower, postoperative.)

B, Phonocardiograms in L. M., showing a reduction in the intensity of the aortic systolic murmur and intensification of the aortic second sound following aortic commissurotomy. (Upper part, preoperative; lower, postoperative.)

Reduction in pressure differential between the left ventricle and the aorta (measured directly during surgery) are shown in Fig. 28, *A-D*. Unfortunately, we have not performed left heart catheterization in any postoperative patient as yet.

Radiographic changes in heart size have been relatively minimal. However, since most of these patients still maintain some residual element of aortic stenosis, even if it is only that degree which is related to stiffness of perhaps extensively mobilized cusps, it would seem unreasonable to expect great reduction in the size of the left ventricle.

The phonocardiogram often shows a great reduction in the intensity of the basal systolic murmur and a return of the aortic second sound. However, there is seldom a complete abolition of the murmur (Fig. 29, *A, B*).

The spectrocardiograms[45] taken before and after aortic commissurotomy show a similar favorable alteration (Fig. 30).

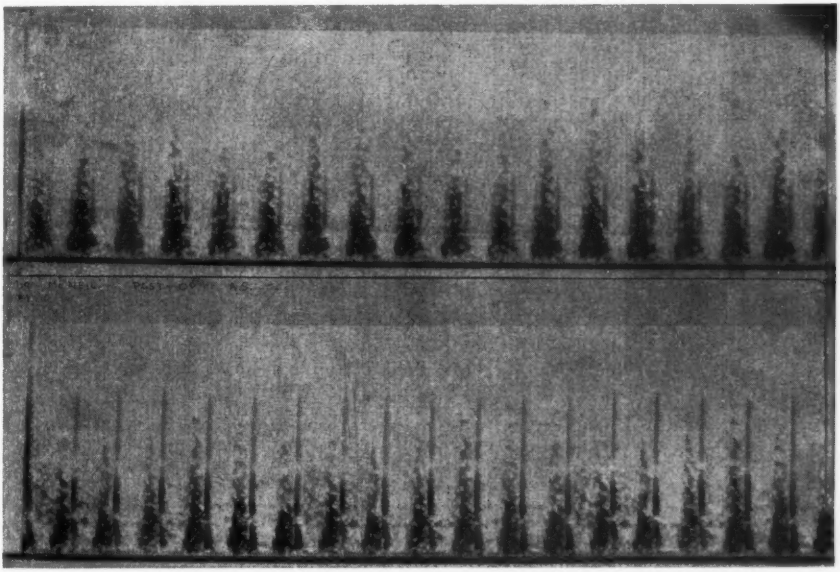


Fig. 30.—Spectrocardiograms showing a reduction in the intensity of the systolic murmur of aortic stenosis and a restoration of the previously imperceptible aortic second sound following aortic commissurotomy, in Patient F. McN. (These tracings were prepared by Dr. George D. Geckeler, Chief of Cardiology at Hahnemann Medical College and Hospital, Philadelphia, Pa.)

SUMMARY

1. A description of the pertinent pathologic and physiologic abnormalities observed in aortic stenosis has been presented.

2. We have detailed the development of concepts and techniques of surgery for the alleviation of aortic stenosis.

3. The indications for and steps in management of patients undergoing aortic commissurotomy are discussed.

4. The improved techniques of management of isolated aortic stenosis and of combined mitral and aortic stenosis are illustrated.

5. The results of our experience to date are epitomized.

6. Trivalvular (mitral, aortic, tricuspid) exploration is recommended by way of a right-sided (third or fourth anterior intercostal space) incision whenever possible in all patients operated upon for a single rheumatic stenotic valve lesion. All correctible lesions found should be treated at the same operative session.

CONCLUSIONS

Severe rheumatic aortic stenosis is a medically intractable and often a promptly fatal illness. However, some patients may survive for years, although with increasing incapacitation. This lesion is reasonably responsive to appropriate surgical separation of the obliterated commissures.

This should be performed, whenever feasible, by the transaortic route, using digital "vision" to explore the valve and to control the course of any instrumentation. Splitting of one or more fused commissures by simple finger pressure is possible in almost one-half of the patients. Instrumental aid is mandatory in the rest.

Coexisting mitral and tricuspid stenosis may be approached and relieved satisfactorily from the right side through the same right anterior thoracic incision. Indeed, it is now our custom to perform elective surgery for "isolated" mitral stenosis by the right-sided thoracic approach largely in order that any other valvular conditions unexpectedly encountered may be treated simultaneously.

The operative mortality and the clinical improvement obtained in surgery for aortic stenosis now closely approach the satisfactory levels established in commissurotomy for mitral stenosis.

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Discussion

DR. HARRISON BLACK, Boston, Mass.—I should like to congratulate Dr. Bailey on his large series but would like to go back to the discussion on aortic stenosis where we started. Our interest on Dr. Harken's services in Boston has been directed, during the developmental phases of aortic surgery, toward determining which patients need surgical relief. I believe that as experience increases one comes to realize that one swallow does not make a summer and that a murmur and thrill (caused by turbulence of blood) do not necessarily indicate a hemodynamically significant valvular lesion. Therefore, up to the present we have chosen to operate only when aortic stenosis has produced part or all of the triad—angina, syncope, or left ventricular failure. When mitral and aortic stenosis have coexisted, we have not corrected the aortic lesion unless the transaortic gradient measured on the table exceeded 50 mm. Hg. (slide) Our choice of this figure is arbitrary but is based on the pressure-work studies of Dr. Richard Gorlin of our Medical Department. (Bull. of New England Med. Center **16**: 13-23, 1954.)

On the diagram you will note that the work of the left ventricle rises steeply when the gradient exceeds 50 mm. Hg. In a group of between 30 and 40 patients with such combined lesions but with an aortic gradient below 50 we have performed mitral valvuloplasty only. The follow-up of these patients over several years apparently justifies this decision, for the vast majority have been markedly improved and have been able to return to a normal level of activity. From this we may conclude that some patients with mitral stenosis and an aortic murmur and thrill may do as well without the aortic operation as with it.

(slide) Dr. Gorlin's study has documented another important point that has become increasingly apparent from clinical experience, that is, that the correction of aortic stenosis cannot be accomplished by the creation of insufficiency any more than is true in dealing with the mitral valve. This diagram reveals that with an aortic valve of a given area the work of the left ventricle increases threefold when insufficiency is added to the stenosis.

Our record with the transventricular approach to the pure aortic stenosis is quite similar to Dr. Bailey's with approximately 30 per cent early mortality and less than 50 per cent survival when the late deaths are included. For this reason we have turned to the transaortic approach described by William Swann of Knoxville. This is vastly better tolerated than the transventricular operation since neither pleural cavity need be opened, both lungs are expanded and available for ventilation throughout the procedure and the problems of ventricular cardiotomy (hemorrhage, standstill, and ventricular fibrillation) are avoided. We use a sternal splitting approach and I think Dr. Bailey will be intrigued by the opportunity this offers to operate on four valves at one sitting.

(slide) In our experience the vast majority of stenotic aortic valves are calcified by the time they become symptomatic and are bicuspid due to fusion of the two anterior cusps. Rarely have we seen the type so commonly pictured in pathology texts—those with an equal fusion of all three commissures. At the time of our first transaortic operation the importance of avoiding a fracture through the heavily calcified anterior commissures was not adequately appreciated and the patient died on the table from embolization of calcified particles. In 12 subsequent cases, fracture with the finger or breadknife valvulotome has been limited to the lateral commissures and extended along the aortic wall anteriorly and posteriorly on each side. By this means a double "trap-door" valve is created without risking embolization and without the production of insufficiency. The calcific bars are not broken into but mobilized as a unit.

The only additional death from a transaortic operation occurred in a woman with advanced coronary artery disease in addition to aortic stenosis. In this case, as in several others, the transaortic gradient has been entirely eliminated. As experience is gained with this procedure and its safety established, we will probably accept for operation individuals in whom the gradient is above 30 or even 20 mm. Hg. We are going to have to accept patients earlier for aortic stenosis surgery in order to correct the lesion

before calcification adds to the risk and seriously interferes with the restoration of competent valve function. To do so undoubtedly will depend on a reliable method of measuring the transaortic gradient before operation.

DR. CONRAD LAM, Detroit, Mich.—I should like to show the post-mortem specimens of the aortic valves of 3 patients which show what may happen when a dilator of the Bailey type is inserted and opened.

(slide) This illustrates a technically successful aortic commissurotomy. The patient was near death from aortic stenosis at the time of the operation and, unfortunately, had adhesive pericarditis which made some dissection necessary before the aortic root could be palpated. The heart was in standstill when the transventricular operation was begun. After the dilatation, the heart was easily resuscitated, and the patient lived for four days with a normal blood pressure. Death resulted from the residuals of cerebral anoxia.

(slide) Following dilatation of the aortic valve in this patient, a piece of tissue was found caught in the blades of the dilator. It was feared that this represented one of the cusps, and as the failing heart was massaged, a diastolic thrill indicative of marked aortic insufficiency could be felt. The post-mortem specimen showed that a piece of the valve had been removed.

(slide) This valve was dilated, but because it was of the congenital type, it did not tear or open up in a permanent fashion. Death resulted from a delayed rupture of the left ventricle, apparently at the site of a laceration made by the guide wire as it was inadvertently forced against the opposite ventricular wall. A valvotomy of the Brock type would have been indicated for this valve. Probably it could have been accomplished with a cutting instrument such as the Bailey mitral guillotine.

DR. WILLIAM SWANN, Knoxville, Tenn.—We would like to show a slide which is now almost two years old. It was previously shown before this Association one year ago at Montreal in the course of a paper "Intracardiac Surgery with the Aid of Artificial Operative Tunnels." When we showed this slide, we described both finger fracture and instrumental incision of the aortic commissures. We believe this was the first time that this operative technique in relation to the aortic valve had been described.

The first patient was operated upon in June, 1953, and was a patient upon whom Dr. Gerbode had previously operated for mitral stenosis. When we operated on the first patient, we knew that Dr. Bailey had, two months earlier, already used the dilator method from above, through a pericardial pouch.

The actual opening through the fused aortic commissures either with the finger or the knife would seem to be the preferable method. This has been graphically illustrated by McMillan of London with motion pictures of the valve before and after the use of the dilator, and before and after accurate incision with the knife.

(slide) This slide illustrates our present preference, the guillotine knife, and also shows the single channel operative tunnel presently used by us. It is fashioned in two layers. There is an inner layer of plastic for ease of maneuver and an outer layer of cotton cloth to lend strength and particularly to aid in hemostasis at the suture line. As to the substance that is used for the auricular appendage substitute, that is a matter of personal preference. We have, at the invitation of Dr. Bailey, been privileged to observe his use of the pericardial pouch. In his hands this method is satisfactory. In our hands the method is unnecessarily time consuming, difficult, and not entirely dependable. Certainly there is no advantage to fashioning the operative tunnel, or pouch, from living tissue inasmuch as it is to be discarded at the end of the procedure. A pouch made of rubber or plastic alone is not satisfactory. Dangerous leakage occurs at the suture line. A fabric such as cotton cloth should be incorporated, at least at the line of suture to the aorta.

DR. I. M. MADOFF, Boston, Mass.—I am certain that the technique which Dr. Swann has described and Dr. Bailey has so successfully carried forward will eventually prove to be an extremely useful procedure. However, with the thought that some

versatility in the technical approach to the treatment of aortic stenosis might produce better over-all results, I would like to review briefly an approach in which we have been interested. This approach is derived from a study of freshly autopsied heart specimens and experiments on anesthetized dogs. As you know, the aortic valve lies in relationship to the left atrium and the right atrium posteriorly and laterally. By inserting the finger into the left atrium, as one does in operating for mitral stenosis, and invaginating the wall of the right atrium at the same time, one can manipulate and compress the aortic valve ring and indirectly change the contour of the valve leaflets. In some instances of aortic valvular stenosis, we have noted that, despite adequate dilatation of the stenotic valve by various instruments, the valve lumen remains the same even though the commissures appear to be separated. However, by compressing the valve ring externally in this manner, the valve ring becomes more mobile and the size of the valve lumen increases. This maneuver will produce an increase in blood flow through the aortic valve area, not only by means of increasing mobility of the valve ring, but also by increasing the size of the valve lumen.

(slide) This shows a typical aortic valvular stenosis with calcification.

(slide) The finger is introduced into the left atrium, the right atrial appendage is inverted and you notice that the valve ring is compressed.

(slide) At the completion of the maneuver this shows the dilated valve orifice to be much larger.

(slide) Since one may potentially further narrow the valve lumen temporarily by compression of the valve ring this instrument, called a flowtome, has been designed. This permits blood flow through the aortic valve at least sufficient for coronary circulation while the compression maneuver is being accomplished. The instrument is introduced through the left atrium and its head is brought out through the aortic valve.

(slide) This is the flowtome in situ prior to compression.

(slide) This shows the compression maneuver with the flowtome in position and how it can permit continuous blood flow.

(slide) At the completion of compression you will notice the significantly dilated aortic valve lumen.

Some fifty heart specimens with aortic stenosis have been examined; in these, this procedure has produced effective aortic valve dilatation in 3 out of 4 cases. However, it can be accomplished without many of the recognized hazards of the other surgical approaches to this lesion. If the procedure is found to be not effective as determined by pressure gradients observed at operation, in a given case, one may proceed with one of the other available methods.

DR. CHARLES P. BAILEY (Closing).—I want to thank the discussants and will try to answer their questions. I well remember previous discussions with Dr. Harken on the subject of aortic stenosis and possible suitable surgery. I am sorry he and Dr. Black did not choose to follow my lead until Dr. Swann followed it and reported corroboration.

I know that abnormal auscultatory findings over the aorta are not necessarily indicative of physiologic abnormality but, on the other hand, they may be. I would like to show the first slide to clarify what I meant when I said there might be a difference between the differential in systolic pressures obtained between the aorta and the left ventricle, preoperatively, and those in the same case obtained directly during operation. You see the marked reduction in the differential pressures, probably because of diminution in the cardiac stroke output, due perhaps to anesthesia, the open chest, and in part to blood loss. Also, after the obstruction of a mitral valve is opened in a case of combined aortic and mitral stenosis there might be difficulty in showing a significant gradient physiologically since the curves may be "damped" due to anesthesia or to the depression of surgery. By left and right combined heart catheterization usually one can tell if the aortic stenosis is significant preoperatively. I think it has become fairly evident by now that if one does not relieve coexisting aortic stenosis when a stenotic mitral valve is opened, he does the patient great disservice, because there is then obstruction of the

outflow valve of the left ventricle but no longer of the inflow. The patient therefore has essentially isolated aortic stenosis, which is a much more malignant disease than is the combination of the two stenotic lesions. I think, however, it would be good to have evidence of a pressure differential of at least 30 mm. before opening the aortic wall to palpate the aortic valve through some kind of pouch.

We do not agree that all aortic valves have become bivalvular when they are calcific. We know that less than one-half of them are bivalvular when they are calcific, and we think that at least 90 per cent of aortic valves operated upon for stenosis are calcific at the time of surgery.

Dr. Lam showed rather beautifully in a few cases how transventricular aortic dilatation can help the valve, and in one how a technical complication can produce insufficiency and cause death of the patient.

(slide) This slide indicates why we gave up the transventricular route to the aortic valve and, of course, the inadvertent creation of regurgitation was one of the reasons. This is a blind, purely instrumental, operation. Since apparently a finger is necessary for the accurate splitting of the commissures, the supravalvular approach certainly is preferable.

Dr. Swann evidently used the guillotine knife before we did, although I am sure we had split the valve digitally as well as dilated it with an instrument prior to that time. The suggestion that a pouch might be used to enter the aortic lumen had been made by Dr. William Glenn prior to the time we used it. He actually used it in experimental animals.

(slide) This slide shows the cause of death in 19 patients operated on by the transventricular route. I think you can see that most of these deaths are related to going through the ventricle which may be very soft, so that there may be uncontrollable hemorrhage, and very irritable so that there may be ventricular fibrillation or cardiac arrest. Either of these rhythmic changes sometimes may be related to the inadvertent creation of aortic insufficiency.

Dr. Madoff's operation is very interesting and, of course, might prove in some cases to be a very great help.

MANAGEMENT OF THE POSTRESECTION SPACE IN TUBERCULOSIS

II. Following Lobectomy

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THE propensity with which tuberculosis affects the lung apex, whether postural, biochemical, or inherent, has directed the efforts of resection surgery primarily toward the upper lobes. Thus, upper lobectomy has become a relatively common operation in the repertory of the thoracic surgeon. In patients without evidence of endobronchial disease or resistant tubercle bacilli, lobectomy was proved to be a safe and often curative procedure. Current reports.[1-6] including our own experience with segmental and subsegmental excision, indicate a definitely higher nontuberculous morbidity than is found with lobectomy.

We shall not discuss the indications for lobectomy, nor the relative function-sparing advantage of saving the right anterior segment or lingula on the left. Rather, we are concerned with the problem of a neutral or positive pressure postlobectomy space and the success of right middle and lower lobe remnants in filling the hemithorax.

The elimination of residual intrathoracic space is not entirely accomplished by expansion of adjacent parenchyma. Relative shifting of the superior mediastinum (Fig. 1), narrowing of the upper interspaces, and a temporary rise in the hemidiaphragm, all serve to reduce the volume requirement of the upper chest. Moreover, the resumption of space relationships by middle and lower lobes is not always an acute process. Upper lobes, harboring enough tuberculosis to require their complete excision, vary widely in size. At operation, one may encounter a range from normal volume to a tiny shrunken paramediastinal wedge occupying one-eighth or less of the hemithorax (Fig. 2). As the upper lobe progressively contracts, even with chest wall and mediastinal compensation, some expansion of middle and lower lobe tissue takes place. Commonly, the right middle lobe or lingula, and the superior segment of the lower lobe, are the units contributing most to this volume readjustment.

Turning from the mechanical considerations, the physiological adjustment, subsequent to lobectomy, may also be minimal. This, in part, is due to the fact that essentially functionless tissue is removed. In the diseased area, although ventilation is reduced less than circulation and oxygen uptake, the net change reflected in pulmonary function studies after full recovery may differ little from preoperative values.[7-9]

From the Veterans Administration Hospital, Sunmount, N. Y.
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MATERIAL

During the five-year period, Jan. 1, 1950, to Dec. 31, 1954, 135 lobectomies were performed at the Veterans Administration Hospital, Sunmount, N. Y. (Table I). A review of this material discloses 30 cases (22 per cent) of post-lobectomy space requiring clinical recognition, and, in most instances, supplementary treatment. We have included in this group cases in which superior segmental resection or wedges from middle or lower lobes was accomplished concurrently with the upper lobectomy. Superior division resection on the left, although the counterpart of a right upper lobectomy, was not included in this series.

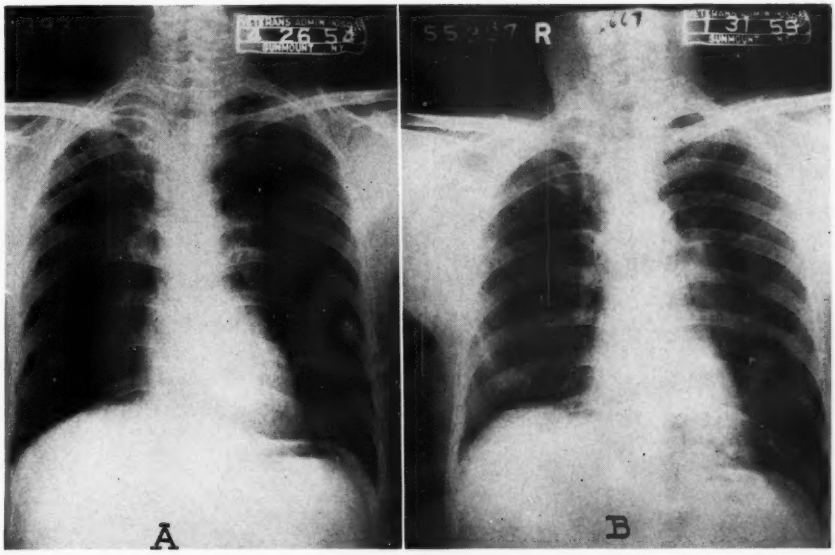

Fig. 1.—*A*, Preoperation. *B*, 8 months postoperative, right upper lobe.

TABLE I. POSTRESECTION SPACES FOLLOWING LOBECTOMY—135 OPERATIONS JANUARY, 1950, TO DECEMBER, 1954

NUMBER OF PATIENTS	CAVITY STATUS PREOPERATIVELY		SPUTUM BACTERIOLOGY PREOPERATIVELY		LOCATION OF SPACE		DATE OF SPACE APPEARANCE POSTOPERATIVELY	
30	Closed	10	Negative	21	Apical pneumothorax	18	1st week	26
					Apical hemothorax	2	2nd week	2
	Open	20	Positive	9	Basal pneumothorax	5	3rd week	1
					Combined (apical and basal pneumothorax)	3	4th week	1
					Apical intrapleural space communicating with subscapular	2		

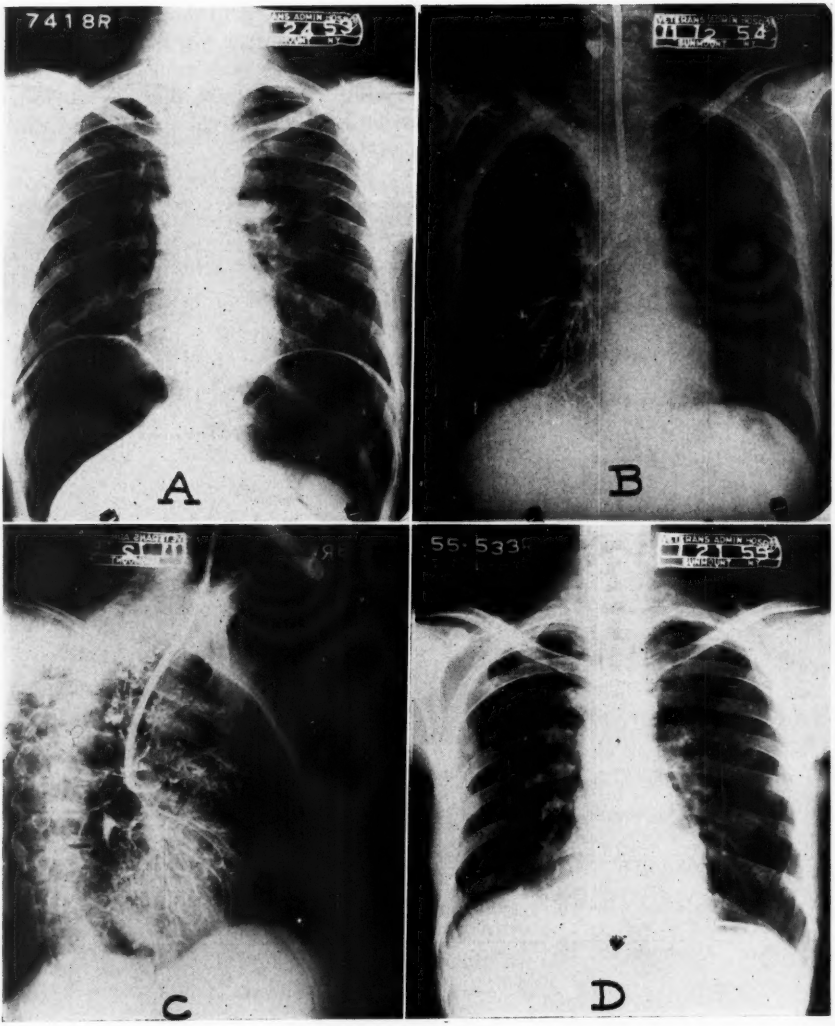

Fig. 2.—*A*, Preoperation. *B* and *C*, Bronchograms. *D*, Postoperative, right upper lobe.

The addition of segmental or subsegmental excision to lobectomy introduces the factors of complications incident to this type of operation. The occurrence of postoperative air leaks may parallel the number of segmental stumps and absolute amount of raw lung surface remaining after such parenchymal barnstorming.

Segments or subsegments from the lower lobe also influence the location of the postresection space (Table I). Air leaks from these areas are prone to result in lateral or supradiaphragmatic pneumothoraces. All basal spaces in our group occurred in cases where segmental or multiple subsegmental excision was added to the upper lobectomy.

The lobes removed in this series represent a variety of moderately and far-advanced tuberculosis, with a cavitary area existing or suspected in the majority. The sputum was almost equally divided between the positive and negative states. As with segmental resections, the preoperative disease state was not found to be related to the frequency of postresection nontuberculous complications.

OPERATIVE MANAGEMENT

Anticipation of a space problem begins at the operating table. An upper lobe of subnormal volume simplifies the problem, as compensation has already begun in adjacent lobes. The physical appearance of the remaining lung is most important. Slow inflation, trapping, or delayed emptying, or any aberrations in ventilation, may foretell difficulties in postoperative expansion. The extent of distribution of residual disease is significant. Fine seeding throughout the parenchyma, stellate areas of fibrosis, with surrounding patchy areas of obstructive emphysema, complicate re-expansion.

Initially, we felt great trepidation in expecting prompt re-expansion of lower lobes containing moderate amounts of residual disease. These were cases in which so-called compromise resection surgery was carried out. The cavity-bearing upper lobe, with adjacent nodular disease, might represent 60 to 70 per cent of the total disease in that lung. The presence of contralateral disease, and other considerations, made retention of the lower lobe or basilar segments almost a necessity if resection was at all feasible. We were most pleased to observe prompt expansion and occupation of almost the entire hemithorax by lower lobes or basilar segments alone which contained various amounts of nodular disease (Fig. 3).

Following the surgeon's estimate of predictable space filling, several measures are available to aid this readjustment. These measures may be divided into two groups; those accomplished within the chest and those applied to the chest wall. Several thoracic surgeons, following the suggestions of Blades, as an adjunct in the Blalock operation, divide the inferior pulmonary ligament to allow ascent of the hilum. This appears to be a useful addition, particularly after left upper lobectomy, but it is doubtful that it is routinely necessary. Any effort to paralyze the hemidiaphragm temporarily is mentioned only to be deplored. One of the main problems in the postoperative period is the proper raising of secretions. Paradox of this muscle not only interferes with ventilation, but robs the dependent lower lobe of the pistonlike action which is vital for an

effective cough mechanism. Far better, from the physiologic standpoint, is the institution of a pneumoperitoneum postoperatively, when it is desired to raise the level of the diaphragm.

The creation of a so-called apical pleural tent, as described by Miscall,[10] is a valuable adjunct. The fashioning of a satisfactory tent, of course, depends upon the integrity of the parietal pleura. Any entry into the extrapleural plane, during mobilization of the upper lobe, may make such a tent impossible. In addition, a rent in the extrapleural or subcostal tissues mitigates against concomitant freeing of the subcostal or extraperiosteal tissues, or against any variation of thoracoplasty. Such openings, even when sutured, may open during the stress of local paradox, allowing the intrapleural and subscapular spaces free communication. In the event of a subsequent bronchopleural fistula, with pleural infection, this sepsis, either pyogenic or tuberculous, may involve the subscapular tissues.

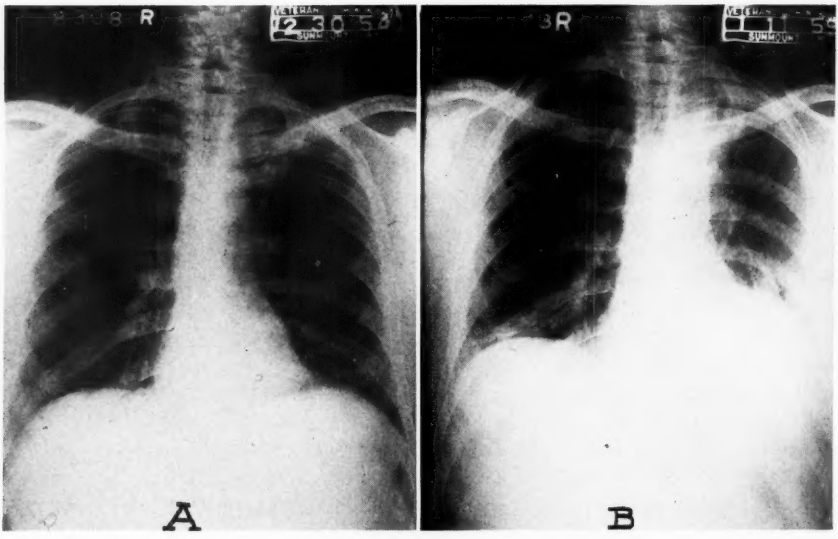

Fig. 3.—*A*, Preoperation. *B*, 6 days postoperative, left upper lobe and superior segment, left lower lobe.

An apical pleural tent may aid in pleuralizing and thus sealing raw lung surfaces of the middle or lower lobes. This same advantage may be secured by mobilization of the subcostal tissues as used by Chamberlain and associates,[1] without removing or altering the adjacent ribs. In this situation, some surgeons have introduced a small plombage into the subcostal space. Our experience with 4 cases of concomitant plombage have been uniformly poor. Each patient became infected, and required subsequent removal and completion of thoracoplasty. In view of the 25 per cent incidence of tuberculous infection, reported by Gibbons, Desforges, and Strieder,[11] in formal plombage with presumably intact extraperiosteal tissues, we believe the insertion of a concomitant foreign body at the time of resection is unwise.[12]

Various modifications of conventional thoracoplasty are in current use. Steele and co-workers[2] have described the division of the upper ribs into short segments without removal as an aid to reduce paradox. Björk's[13] description of a concomitant osteoplastic thoracoplasty may be a most valuable procedure when a simultaneous maneuver is necessary to reduce intrathoracic space.

Our modifications and indications for the pre- and postresection thoracoplasty and the pleural tent, each comprising a group of approximately 25 cases, will be discussed in the next and last study on the management of the postresection space in tuberculosis.

TREATMENT

Assuming that no type of concomitant procedure was carried out with the resection, the treatment of a postoperative pneumothorax is contingent upon the degree of air leak, if any, contributing to the space.

The majority of space problems may be divided into two groups. In the first are apical and posterolateral caps, which on single or multiple aspirations demonstrate a neutral or slightly negative pressure relationship. Patients in this category usually have no overt symptoms of a fistula; i.e., absence of irritative cough, streaking, or fever. Moreover, the surgeon may, as a result of palpation or the behavior of the lung during operation, have reason to anticipate delay in re-expansion of the parenchyma. Retention of secretions or excessive air leaks in a moderately emphysematous lung, during the postoperative period, contribute to failure of space occupation. If unassociated with a hemothorax, such a space may not require any definitive treatment. Serial films may disclose an ever-decreasing air cap. One may expect gradual expansion of the remaining lung and the eventual compensation of superior mediastinum, upper interspaces, and diaphragm, to reduce the hemithorax and eliminate any residual pneumothorax (Fig. 4). Uncertainly as to the presence or absence of a bronchial communication, if not resolved with manometry, may frequently be clarified by bronchography.[14]

Apical hematomas of any magnitude (two or more interspaces) are best evacuated by secondary thoracotomy. At this time, a concomitant chest wall procedure may be added to reduce the actual or potentially infected space. Other than the small, so-called rim hemothorax, which may undergo spontaneous resolution, we have not been successful even with the assiduous use of enzymes in evacuating such collections of blood through a needle. Occasionally, with the use of an intercostal catheter, clearing of the space has been accomplished. However, an appreciable collection of clotted blood at the apex, with possible intermittent fistulization, is ripe for secondary or primary tuberculous infection. Such an apical empyema may be anticipated in some cases and surgically prevented.

The presence of a positive tension space in the postoperative period requires immediate treatment. Usually such a pneumothorax is manifest shortly after removal of the primary thoracotomy tubes. An x-ray at this time may show a so-called blowing up of the upper hemithorax with concave depression of the lower lung and shifting of the superior mediastinum to the contralateral side.

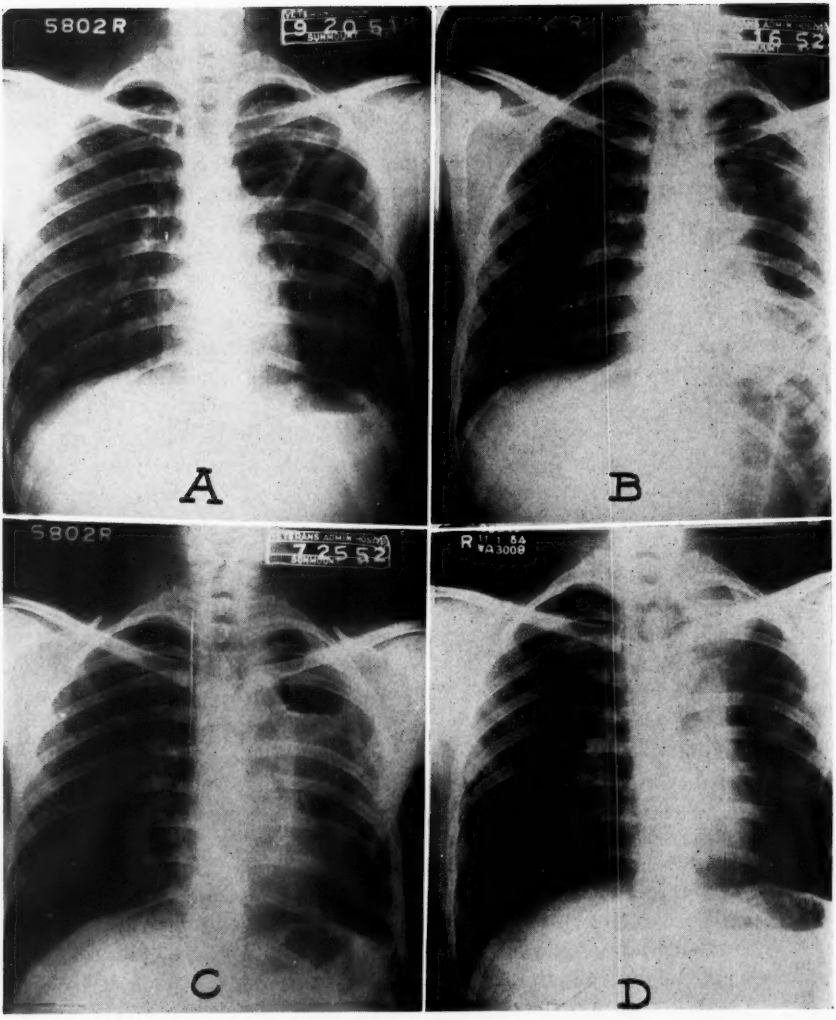

Fig. 4.—*A*, Preoperation. *B*, Postoperative, left upper lobe, showing left apical space. *C*, Persistent left pneumothorax 3 months postoperative. No treatment, no fistula demonstrated. *D*, Two and one-half years postoperative. Marked compensatory shifting of superior mediastinum.

The surgeon may now elect the following alternatives of treatment. Prompt intubation of the space will relieve any symptoms associated with the local tension pneumothorax. Closure of an underlying fistula of any magnitude usually will not occur, however, with tube drainage alone. The necessity of a space obliterating procedure for the inevitable empyema, in addition to the possibility of closing the fistula, may make the postresection thoracoplasty a logical step. Since accurate intubation of a subthoracoplasty space is difficult, prior insertion of a tube is of value. The tube may be maintained postoperatively until there is evidence of fistula closure.

Observation has shown that the presence of an intercostal catheter after approximately one week, even with constant antibiotic coverage, is nearly always associated with secondary infection. The occurrence of a postlobectomy fistula may then present the surgeon with the contrary treatment alternative, that of secondary thoracotomy and attempted revision of the bronchial stump. Since this procedure is much safer when risk of infection is minimal, prior intubation may be omitted.

If direct surgical closure of the fistula is successful, adjacent parenchyma may re-expand and cure is accomplished. Yet even in the absence of a positive space, the remaining lung may be incapable of occupying this area. Anticipation of this problem may lead the surgeon to perform a concomitant thoracoplasty at the time of secondary thoracotomy. Otherwise, chest wall surgery may be required as a third operation.

RESULTS

Review of treatment employed in this group (Table II) discloses that one-third of the patients were managed by intrapleural catheter drainage. These cases represented small transient bronchopleural fistulas, which closed after one to two weeks of underwater suction. The apical spaces in 2 patients required prolonged drainage up to five weeks, and were in each instance complicated by penicillin-resistant hemolytic *Staphylococcus aureus* infection. The fistulas closed satisfactorily; however, since tubercle bacilli were not recovered from the space, thoracoplasty was not added.

Pneumoperitoneum, with or without intubation of the chest, was used in 4 instances. This combination is useful, chiefly for the supradiaphragmatic space so prone to occur when superior segmental excision or multiple wedging of the lower lobe is added to the upper lobectomy.

Our employment of a concomitant thoracoplasty was limited to 2 cases. The first was actually a modified plombage, with the ribs retained and Gelfoam placed in the subcostal space. The subsequent development of an upper lobe stump fistula, which communicated with and infected the subcostal and subscapular spaces, led to removal of the ribs and appropriate drainage. The second case had no complications.

Our indications for postresection thoracoplasty in this group were the persistence of a large apical space, with or without an associated fistula, or the demonstration of a positive culture for tubercle bacilli from this space. The

thoracoplasty as used in this manner was satisfactory in aiding fistula closure, although results were generally better when the thoracoplasty was done over an intrapleural tube which was gradually withdrawn as the fistula healed.

The results of space treatment are noted as cured or satisfactory in 21 patients. The unsatisfactory cases were largely due to the injudicious and prolonged use of catheter drainage alone, when the addition of a small apical thoracoplasty might have hastened or improved the end results.

The current status of tuberculosis in each patient with a postoperative pneumothorax is summarized (Table II).

TABLE II. TREATMENT OF SPACE PROBLEMS FOLLOWING LOBECTOMY (30 CASES)

TREATMENT		DURATION OF INTERCOSTAL DRAINAGE		INFECTION (PREDOMINANT ORGANISM)		RESULTS OF SPACE TREATMENT		STATUS OF TUBERCLE BACILLI (N.T.A.)	
No treatment	7	1 week	7	Hemolytic *Staphylococcus aureus*	2	Cured	12	Inactive	23
Thoracentesis	3	2 weeks	3					Active	3
Intercostal catheter	10	4 weeks	1	Tubercle bacilli	2	Satisfactory	9	Negative (In hospital)	1
		5 weeks	1						
Intercostal catheter with pneumoperitoneum	2	6 weeks	1	Mixed (staphylococcus and tubercle bacilli)	1	Unsatisfactory	9	Deceased (1 tuberculous, 1 nontuberculous)	2
Pneumoperitoneum	2							No follow-up	1
Concomitant thoracoplasty	2								
Postresection thoracoplasty	4								

COMMENT

A local postresection tension pneumothorax requires prompt clinical recognition. This situation, with the implication of a frank bronchial stump fistula, may be a surgical emergency equivalent to a nonsurgical spontaneous tension pneumothorax. Even very small or intermittent fistulization in the tuberculous patient may lead to an eventual pyogenic or tuberculous empyema in a persistent intrathoracic space.

Surgical decompression may follow two electives: multiple needle, or, if positive pressure is apparent, closed thoracotomy intubation of the space. This expedient will eliminate the symptoms of tension. Although open thoracotomy is theoretically always possible even after intubation, the chances of success in fistula closure and local débridement of the space diminish greatly as intubation is prolonged.

Following the diagnosis of a postresection space, if the surgeon has had good experience with fistula revision, suspects the local anatomy of the leak,

and can adequately deal with the space—then this may be his golden moment for performing secondary open thoracotomy.

If these considerations do not apply, and closed drainage has been carried out, an elective postresection thoracoplasty may be the only useful alternative.

SUMMARY

An analysis is presented of 30 cases of postlobectomy space, occurring in a series of 135 lobectomies for tuberculosis. Significant factors in the genesis of this complication are discussed. Measures for both prophylaxis and postoperative management of the postresection apical pneumothorax are suggested.

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LUNG COMPARTMENT DETERMINATION

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WITH the rapid growth of cardiopulmonary surgery there has arisen a great need for a method of determining just what is the true normal value of such factors as the cardiac index, total lung volume, residual capacity, vital capacity, maximum breathing ability, oxygen consumption and removal, etc.

The present methods of predicting the normal values of these function studies for any specific patient are based upon his body surface area, or are assumed to be some average value which has been determined upon a series of healthy subjects. It is hardly necessary to point out that body surface area is a very variable factor in itself, since it depends on the fat mass as well as weight and height. On the other hand, if one uses an "average value" as a predicted value for a specific patient that too is equally variable.

METHOD

The total lung capacity can be calculated from a single inspiratory postero-anterior chest film.

We had a series of clinically normal standing subjects inspire maximally from a recording spirometer and at the point of maximal inspiration an x-ray (posteroanterior) picture was taken. This was then immediately repeated in full expiration, after which it was done in the lateral plane at full inspiration and expiration while breathing into the spirometer. These last films were soon found to be unnecessary.

Residual air determinations were carried out after the roentgenology with consideration of the resting and fasting state. Our modification of the Cournand open-circuit method was used.[1]

CALCULATIONS

The whole thoracic cage is treated mathematically as a paraboloid of revolution with the vertex at the center of a horizontal line joining the highest points of the inner borders of the first ribs and "base" on the horizontal line joining the attachment of the diaphragm to the ribs in the midaxillary lines (Figs. 1 and 2).

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$$V = \frac{II}{2} ab^2 \qquad \text{(Ref. 2)}$$

Where V = volume of paraboloid of revolution of cage in milliliters.
a = distance from vertex to base in centimeters.
b = length of one-half "base" in centimeters.

To obtain the total lung air capacity one must subtract from this the volumes of the "diaphragmatic dome" above the "base line," the heart, the superior mediastinum and adjacent vertebral bodies, the posterior mediastinum and its adjacent vertebral bodies together with the lung parenchymal and blood volume.

The "diaphragmatic dome" is treated as the sum of two half segments of a sphere to allow for differences in the volumes of the right and left leaves (Figs. 1 and 3).

$$V = \frac{II}{6} h^2 (3b - h) \qquad \text{(Ref. 3)}$$

Where V = volume in milliliters of one-half segment of sphere.
b = length in centimeters of "one-half base."
h = height of top of diaphragmatic leaf from base line in centimeters.

This is calculated for each diaphragmatic leaf and the two added together to give the volume of the total diaphragmatic correction.

The heart is considered to be a paraboloid of revolution with vertex at the center of the base of the heart as soon "radiologically" where the heart borders become confluent with the superior mediastinum, usually at the upper border of the seventh rib posteriorly, or the lower border of the third rib anteriorly, and "base" on a horizontal line at the diaphragmatic surface of the heart (where the dome of the left leaf of the diaphragm and the inferior border of the heart merge) (Figs. 1 and 3).

The general formula for a paraboloid of revolution becomes:

$$V = \frac{II}{2} a_2 \frac{(t_1 - t_2)^2}{2}$$

Where V = volume of heart in milliliters.
a_2 = vertical distance from base of heart to level of diaphragmatic dome in centimeters.
t_1 = distance from midline to right border in centimeters.
t_2 = distance from midline to left border of heart in centimeters.

The superior mediastinum and adjacent vertebral bodies are treated as one unit mathematically. Since this is part and parcel of the original thoracic cage paraboloid of revolution, one may visualize it as a parabolic surface in cross section which when multiplied by the width "w" of the superior mediastinum will give its volume in milliliters. The base of this parabola is equal to the distance in centimeters from the midline, at the level of the upper border of the heart, laterally to the inside of the thoracic cage in the midaxillary line labeled b_1 on Figs. 1 and 2.

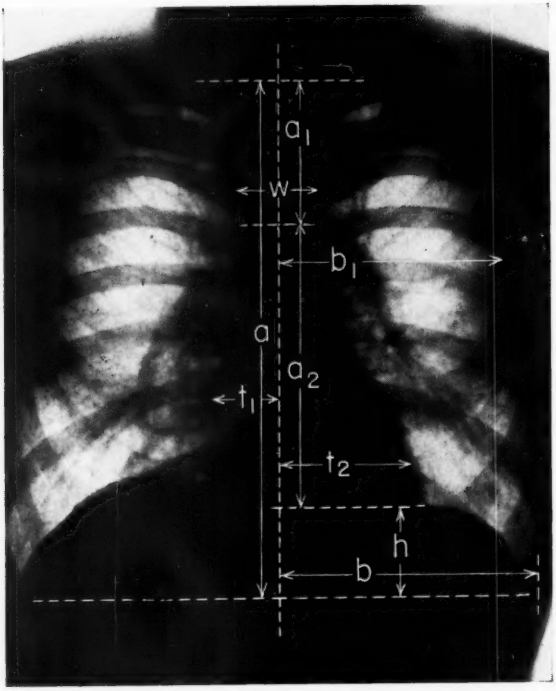

Fig. 1.

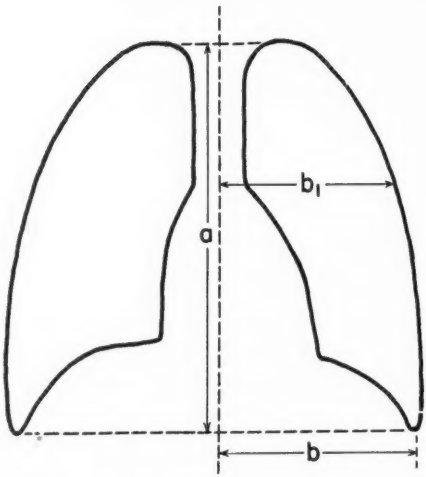

Fig. 2.

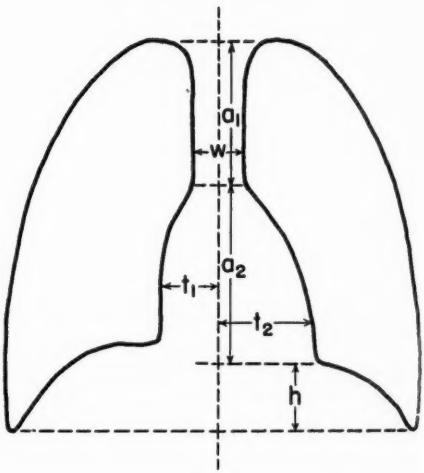

Fig. 3.

The general formula for the area of a section of a parabola is:

$$A = \frac{4}{3} a_1 b_1 \qquad \text{(Ref. 4)}$$

Where A = area in square centimeters of parabolic surface.
a_1 = distance in centimeters from thoracic cage vertex to upper border of heart.
b_1 = distance from mid-point of upper border of heart measured horizontally laterally to inside of ribs.
Now $V = A \cdot w$.
Where V = volume in milliliters of superior mediastinum and adjacent vertebral bodies.
A = area of parabolic cross section in square centimeters.
w = width of superior mediastinum in centimeters.

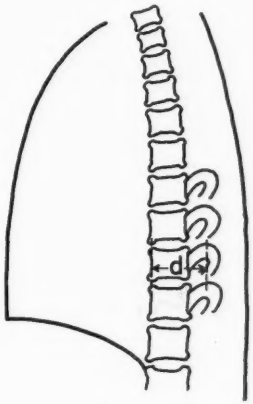

Fig. 4.

The volume of the vertebral bodies adjacent to the posterior mediastinum (which actually is the volume of the remaining vertebral bodies in the "thoracic cage") is obtained by multiplying their width by the length and depth of this column. The width is taken as equal to that of the superior mediastinum. The length is the distance from the upper border of the heart to the "base" of the thoracic cage ($a - a_1$) (Figs. 2 and 3) while the depth d is the distance from the anterior surface of the lower thoracic vertebral bodies to the inner surface of the angles of the ribs at the same level (Fig. 4). One may assume the depth to be 6.1 cm. since there is very little variation in this figure. With this assumption, a lateral film is unnecessary.

In correcting for the volume of the lung parenchyma and its contained blood, our pathology department has found this to vary from 685 ml. to 1,087 ml. We classify the lung volumes into Groups 1 to 5, respectively, 700, 800, 900, 1,000, and 1,100 ml. An average volume is 900 ml. From the x-ray film, one decides to which group the patient belongs and adds 100 ml. to this volume because the x-ray is taken at full inspiration when the output of the right ventricle temporarily is approximately 26 c.c. more than the left per beat so that the in vivo volume is approximately 200 c.c. more than the full expiratory

or cadaveric volume, which would occur about 8 beats later. There is indirect experimental confirmation of this reasoning.[5] It has also been shown by an entirely different method that the volume of the blood contained in the pulmonary vessels, left heart, aorta, and certain large systemic arteries (common and external iliac) is 1,160 ± 246 ml.[6]

TABLE I

PATIENT	AGE AND SEX	VITAL CAPACITY	RESIDUAL CAPACITY (MILLILITERS)	EXPERIMENTAL TOTAL CAPACITY (MILLILITERS)	CALCULATED TOTAL CAPACITY (MILLILITERS)	DIFFERENCE (MILLILITERS)	PER CENT DIFFERENCE
W. I.	41 F	4016	1935	5951	5928	− 23	0.39
G. A.	33 M	4740	940	5680	5720	+ 40	0.75
G. J.	40 M	5300	3320	8620	8557	− 63	0.73
H. R.	25 M	4107	1986	6093	6160	+ 67	1.07
C. J.	51 M	3510	2314	5825	5883	+ 58	0.97
K. G.	19 M	3668	1394	5062	5038	− 24	0.47
M. J.	41 M	4784	1697	6480	6400	− 80	1.18
C. M.	27 M	5276	1237	6513	6557	+ 44	0.67
M. W.	30 M	5480	1685	7165	7170	− 5	0.08
L. A.	31 M	6798	2134	8932	9030	+ 98	1.08
W. F.	47 F	1840	1028	2868	2888	+ 58	0.50
P. S.	28 M	3970	2278	6248	6221	− 27	0.43
R. H.	26 M	5000	1397	6397	6409	+ 12	0.05
W. A.	28 M	5650	1630	7280	7404	+124	1.70
G. H.	34 M	4900	1152	6052	6090	+ 38	0.63
J. H.	34 F	2890	871	3761	3766	− 05	0.01
C. J.	15 M	4620	775	5395	5314	− 89	1.60
S. J.	27 M	3950	1115	5065	5121	+ 56	1.10
R. T.	36 M	3840	1705	5545	5623	+ 78	0.71
M. R.	34 M	4150	910	5060	5070	+ 10	0.05
J. J.	24 M	3240	734	3974	3910	− 64	1.61
D. C.	23 M	5180	1110	6290	6340	+ 50	0.79

TABLE II

PATIENT	CAGE	DIAPHRAGM		SUPERIOR MEDIASTINUM	INFERIOR MEDIASTINUM	LUNG AND BLOOD	HEART
		RIGHT	LEFT				
W. I.	10880	1160	571	700	480	1000	1051
G. A.	12760	636	636	1350	696	1100	1300
G. J.	14860	1475	764	1005	797	1100	965
H. R.	10860	496	496	1045	586	1100	980
C. J.	9250	340	340	695	495	900	600
K. G.	9300	785	785	662	500	900	630
M. J.	11450	2205	900	504	667	900	775
C. M.	11720	1121	520	779	530	1100	1113
M. W.	12600	1275	797	714	555	1200	888
L. A.	15180	1035	923	900	697	1200	1395
W. F.	6120	548	112	465	375	900	821
P. S.	10300	608	410	876	403	1000	881
R. H.	10550	754	544	806	458	900	679
W. A.	13330	1437	579	975	590	1200	1145
G. H.	10420	814	468	732	472	1200	647
J. H.	5770	58	58	560	196	800	332
C. J.	9700	884	652	575	445	900	930
S. J.	11100	975	975	621	420	1000	1040
R. T.	10720	870	535	1022	655	1100	915
M. R.	9450	755	755	550	405	900	880
J. J.	7280	706	360	544	260	800	700
D. C.	11570	800	577	1040	595	1100	1120

RESULTS

The difference between the experimentally observed and calculated values for the total lung capacity was 0.75 per cent over a range or 0.01 per cent to 1.70 per cent in 22 subjects.

Table I is a summary of the results. Table II gives a summary of the components of the calculations used in their derivation.

DISCUSSION

These results show how accurately the total lung capacity can be calculated from the single posteroanterior film.

On our surgical service where all patients are studied in our cardiopulmonary laboratory we repeatedly found that the available predicted normal range was too wide to give us a proper estimate of a patient's deviation from his own normal. Our cardiac cases with grossly increased residual volume (unless they had very large hearts) and diminished vital capacity were especially hard to evaluate.

This method is applicable to all forms of chest pathology. We are now using it in our daily work.

SUMMARY

The vital capacity was determined on a recording spirometer. An x-ray film (posteroanterior) at full inspiration was taken simultaneously. The residual capacity was then determined. Clinically normal subjects were used throughout.

The total lung capacity was calculated by the method outlined from a single posteroanterior x-ray film in full inspiration.

There was an average difference of 0.7 per cent between the experimentally observed and the calculated total lung capacity.

The range of this difference was from 0.01 to 1.75 per cent.

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MEDIASTINAL TUMORS AND CYSTS

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THE present paper is a survey of primary mediastinal tumors and cysts, treated at the Clinic of Thoracic Surgery at Sabbatsberg Hospital during the period 1944 to 1954. As the survey is based on a patho-anatomic classification, we have included only those cases documented with patho-anatomic material.

The following lesions are excluded from this study: (1) carcinoma and benign tumors of the esophagus; (2) mediastinal extension or metastasis of bronchial carcinoma and other metastatic tumors as well; (3) intrathoracic goiter; and (4) systemic diseases and non-neoplastic processes such as leukemia, Hodgkin's disease, tuberculoma, sarcoidosis, aneurysms, and parasitic cysts.

Table I shows the composition of our material in all 155 cases. Some rare tumors and cysts, other than the processes just mentioned, are missing in the material: aberrant parathyroid adenoma and cysts of esophageal and enterogenous origin. We have found no specimens of these kinds among the patients treated at the Clinic in these eleven years. The absence of enterogenous cysts is explained by the fact that children usually are not seen in this Clinic.

TABLE I

Schwannoma and neurofibroma	38
Ganglioneuroma	11
Ganglioneuroblastoma	3
Neurogenic sarcoma	4
Sympathicoblastoma	2
Fibroma	2
Lipoma	1
Fibromyxosarcoma	5
Chondrosarcoma	1
Hemangiomatous tumors	5
Lymphangioma	7
Lymphosarcoma	5
Plasmacytosarcoma	1
Thymoma	19
Thymic cyst	1
Adult teratoma	14
Malignant teratoma	2
Tumor of bronchial adenoma type	3
Bronchial cysts	21
Pericardial cysts	6
Cysts of uncertain origin	4
Total	155

It will be observed, however, that we have taken up 3 cases classed as bronchial adenoma in our material. These were large tumors, wholly localized

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within the mediastinum and without any conspicious anatomic connection with lung, bronchi, or trachea. They presented the typical microscopic structure of bronchial adenoma. We have not been able to find any similar cases in the literature. We believe these tumors are related to the teratomas.

In order to determine whether the percentage distribution of the various diagnoses coincides with that found in other homogeneous series, we have made a comparison, as shown in Table II. In this Table, we have excluded from the compared series, all cases not included in our series. It will be seen that there is considerable statistical incongruity between the distributions in the different series. If they are summed up, however, the distribution in the combined material of 241 cases tallies rather well with that in ours. The similarity is good in some of the larger groups, i.e., benign neurogenic tumors and bronchiogenic cysts. Teratomas, on the other hand, are less frequent in our series than the combined material, partly due to the astonishingly high frequency of these specimens in the material of Curreri and Gale.[14] Thymoma is more frequent in our material. We have rather few pericardial cysts but rather many lymphangiomas. It is possible that some pericardial cysts in the earlier authors' materials in reality have been lymphangiomas. These two cystic affections are rather difficult to separate.

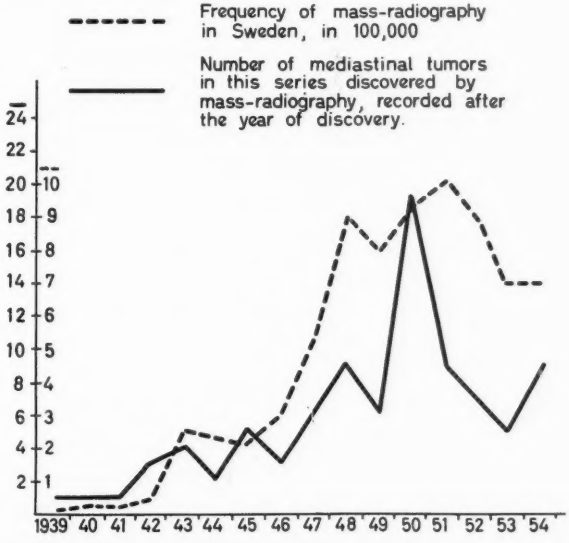


Fig. 1.—Annual distribution of mediastinal tumors discovered by mass radiography in this series compared with the frequency of mass radiography in Sweden.

Before we proceed with a description of the different groups of material, we want to stress the importance of mass radiography for the discovery of these mediastinal affections. It is, indeed, reasonable to say that the extensive use of mass radiography on the Swedish population during the last decade, has been responsible for detection of most of this material. In about 95 out of our 155 cases (about 61 per cent), the mediastinal affection was discovered by

TABLE II

TYPE	BLADES[7]	BRADFORD, MAHON, AND GROW,[9] 1947	BREWER AND DOLLEY,[10] 1949	CONKLIN,[13] 1950	CURRERI AND GALE,[14] 1949	SHIDLER AND HOLMAN,[49] 1952	TOTAL	PER CENT OF TOTAL	OUR SERIES	PER CENT OF OUR SERIES
Benign neurogenic	29	6	16	5	6	15	77	32.0	52	33.5
Malignant neurogenic	1	0	1	0	1	0	3	1.2	6	3.9
Fibroma	1	0	0	2	0	1	4	1.6	2	1.3
Lipoma	3	4	0	0	1	0	8	3.3	1	0.6
Connective tissue sarcomas	0	0	4	1	0	0	5	2.1	6	3.9
Hemangioma	0	0	0	0	0	0	0	0.0	5	3.2
Lymphangioma	0	1	1	1	1	0	4	1.6	7	4.5
Lymphosarcoma	2	0	0	4	1	0	7	2.9	5	3.2
Plasmacytosarcoma	0	0	0	0	0	0	0	0.0	1	0.6
Thymoma	6	3	2	0	2	4	17	7.5	19	12.3
Thymic cyst	0	0	0	0	0	0	0	0.0	1	0.6
Esophageal cysts	0	0	0	0	1	0	1	0.2	0	0.0
Pericardial cysts	10	7	2	2	2	2	25	10.4	6	3.9
Bronchiogenic cysts	23	6	3	4	2	2	40	16.7	21	13.6
Bronchial adenoma	0	0	0	0	0	0	0	0.0	3	2.0
Benign teratoma	14	4	8	5	11	1	43	17.8	14	9.0
Malignant teratoma	6	0	0	0	1	0	7	2.9	2	1.3
Undefined cysts	0	0	0	0	0	0	0	0.0	4	2.6
Total	95	31	37	24	29	25	241	100.0	155	100.0

routine mass radiography. The percentage is about 68 for the benign and 23 for the malignant growths. Fig. 1 gives the distribution of the cases discovered by mass radiography in Sweden as to the year of discovery (full drawn line), and the frequency of mass radiography in Sweden during these years (in hundred thousands; dotted line). A rather close agreement can be noted between the frequency of mass radiography on the one hand and the number of cases discovered by this method, on the other hand.

The specific sites of origin of the various mediastinal tumors and cysts in our series are shown in Table III. There is a definite similarity between the dispersion of the different kinds of tumors in our material and the surveys of localization given by Schlumberger,[44] and Hochberg and associates,[27] among others.

TABLE III

Anterior Superior Mediastinum.—		*Posterior Superior Mediastinum.*—	
Thymoma	16	Schwannoma and neurofibroma	24
Adult teratoma	10	Ganglioneuroma	6
Malignant teratoma	2	Malignant neurogenic tumors	2
Lymphosarcoma	5	Bronchial cysts	6
Fibroma	2	Adult teratoma	1
Lipoma	1	Hemangioma	1
Fibrosarcoma	1	Plasmacytosarcoma	1
Hemangioma	4	Chondrosarcoma	1
Lymphangioma	2	Total	42
Bronchial adenoma	2		
Bronchial cysts	4	*Posterior Inferior Mediastinum.*—	
Thymic cysts	1	Schwannoma	14
Undefined cysts	4	Ganglioneuroma	8
Total	54	Malignant neurogenic tumors	4
		Fibrosarcoma	3
Hilar Region.—		Lymphangioma	4
Bronchial cysts	6	Bronchial cysts	5
Adult teratoma	3	Bronchial adenoma	1
Thymoma	3	Total	39
Lymphangioma	1		
Total	13		
Anterior Inferior Mediastinum.—			
Fibrosarcoma	1		
Pericardial cysts	6		
Total	7		

NEUROGENIC TUMORS

These are the most frequent among the mediastinal tumors. According to Schlumberger,[44] reports of at least 230 cases have been published up to 1951, Hirschfeld,[26] however, gives the figure as 172 cases reported up to the same year.

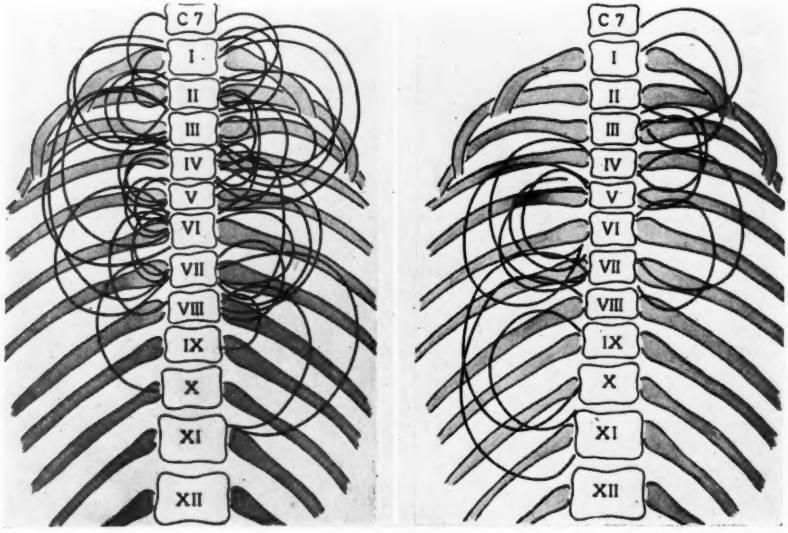
Histologically, they are classed as benign tumors: schwannoma (neurinoma), neurofibroma, and ganglioneuroma and malignant tumors: neurogenic sarcoma (malignant schwannoma), and sympathicoblastoma. There is also a tumor called ganglioneuroblastoma, which microscopically, seems to be intermediate between a ganglioneuroma and a sympathicoblastoma.

Of these tumors, schwannoma and neurofibroma are the most common, followed by ganglioneuroma. According to Rodriguez and co-workers,[42] 90 cases of mediastinal ganglioneuroma had been published up to 1951.

According to Ackerman and Taylor,[1] 14.6 per cent of all mediastinal neurogenic tumors are malignant, but Brewer and Dolley[10] give a figure as high as 33 per cent. Within the malignant groups, malignant schwannoma seems to be most frequent. Hollingsworth[28] (1946), however, collected 18 cases of mediastinal sympathicoblastoma from the literature and added 1 of his own, Davis[15] (1947) found 14 cases in the literature and presented 2 of his own cases.

A comparison of the frequency in Ackerman and Taylor's[1] homogeneous material of 48 mediastinal neurogenous tumors, and in ours of 58 cases, follows:

	ACKERMAN AND TAYLOR	OURS
Neurinoma and neurofibroma	27	38
Ganglioneuroma	7	11
Ganglioneuroblastoma	7	3
Neurogenic sarcoma	4	4
Sympathicoblastoma	3	2
	48	58



A. *B.*

Fig. 2.—Location and approximate size of: *A*, 38 neurinomas and neurofibromas, and *B*, of 14 ganglioneuromas and ganglioneuroblastomas.

As seen, the proportion of clearly malignant tumors is lower in our series than in Ackerman and Taylor's material (10.3 per cent to 14.6 per cent) and the proportion of ganglioneuroblastomas is lower in ours also. This is probably due to the fact that the average age in these tumor groups is low, as children are infrequently seen in our Clinic.

The *benign neurogenic tumors* (including ganglioneuroblastomas) are all similar as to site and gross anatomy. They are invariably located at the costovertebral sulcus, growing close to the lateral surface of the columna and the most proximal part of the ribs. They arise subpleurally and bulge into the

thoracic cavity with a pleural covering. They are always well encapsulated. The location in this series varies from the level of the seventh cervical vertebra to a point just above the diaphragm, i.e., the level of the tenth thoracic vertebra (Fig. 2). The distribution on both sides is about even (24 cases on the left side, 27 cases on the right side, and 1 case was bilateral). The size varies from that of a walnut to that of a large grapefruit. They are spherical or ovoid in shape with a smooth pleural surface. Their tissue is mostly solid, sometimes with degenerative cysts, grayish-white often yellow-spotted. The surgeons often report these tumors as being connected with the sympathetic cord and/or the spinal nerves (25 per cent and 63 per cent, respectively). In about 20 per cent of the cases, the tumor tissue shows an extension into one or several intervertebral foramina. The x-ray picture of dilated foramina is an important diagnostic sign in these cases (Fig. 3)!

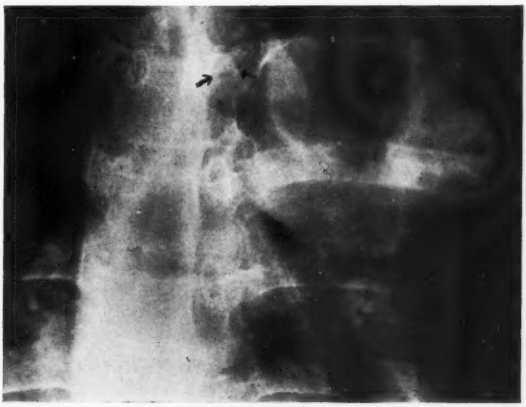
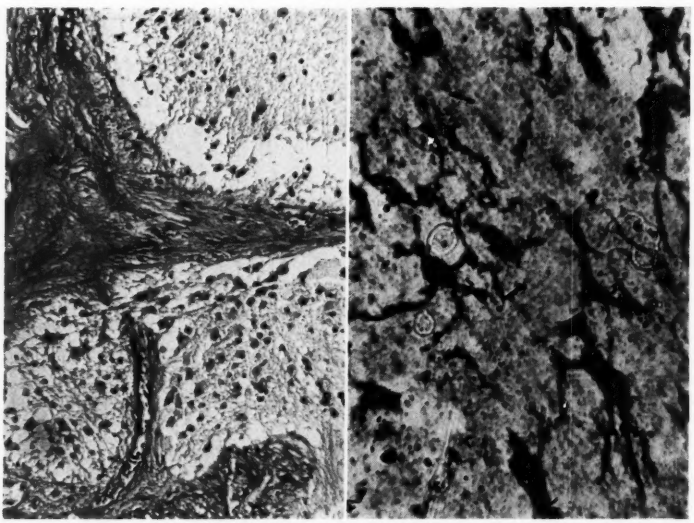


Fig. 3.—Roentgenogram of a dilated intervertebral foramen in a case of neurinoma with interforaminal extension. The area designated by the two arrows shows the absence of normal bone architecture.

In our series, the tumor has presented the gross picture just described whether the microscopic picture was that of schwannoma, combined schwannoma-neurofibroma, ganglioneuroma, or ganglioneuroblastoma. In only 2 cases, with a pure neurofibromatous histologic picture, has the gross picture been atypical; sausage-shaped, markedly gelatinous tumors. In 1 of these cases, the tumor was a local manifestation of a generalized von Recklinghausen's disease. Multiple small nodules along the intercostal nerves were additionally observed.

The histologic picture in 36 of these tumors has been that of schwannoma, either pure, or in a varying degree combined with neurofibromatous structures. As many of the tumors present regressive changes with abundant formation of hyaline fibrous tissue, we find it difficult to draw the border line between schwannoma and neurofibroma. However, in another 2 cases, the picture was that of a pure neurofibroma without appreciable schwannoma-pattern and also without regressive changes. These 2 cases presented an atypical gross picture, as described before.

Eleven of the tumors were mature ganglioneuromas and 3 were tumors with a less mature structure namely, ganglioneuroblastomas. The dominating tissue in all these neoplasms is neurofibromatous, most often consisting of a network of bundles separated by a loose connective tissue. The bundles contain Schwann cells, rests of myelin sheets, and sometimes demonstrable axons. Nerve cells appear scattered or in small nests. In the ganglioneuromas, the nerve cells are of a mature type and often sparse. The ganglioneuroblastomas are rich in nerve cells of an immature type, varying in size, often multinucleated. The nests of these cells are surrounded by a peculiar loose, cobwebby tissue, rather similar to a glial network. Some of the fibers in this tissue seem to be axons. With a combined gold-silver-impregnation method, we have also been able to demonstrate glia cells often of an immature uni- or bipolar type in these islands (Fig. 4).



A. *B.*

Fig. 4.—Ganglioneuroblastoma. *A*, Nests of nerve cells surrounded by a cobwebby tissue. (van Gieson stain, ×120.) *B*, Glia cells in the cobwebby tissue. (Silver-gold impregnation, ×560.)

The incidence of females, in our series and Ackerman and Taylor's[1] series of benign neurogenic tumors and ganglioneuroblastoma, is the same, 60 per cent. The age distribution is different for the various histologic groups. Thus, for the schwannoma-neurofibroma group the mean age at operation is 43, for ganglioneuromas, 25, and for ganglioneuroblastoma 6 years and 7 months. The corresponding figures in Ackerman and Taylor's[1] series are 43, 14, and 6 years, 5 months, respectively.

These benign tumors grow slowly and often attain a considerable size without giving any symptoms. Thus, 36 of the 52 patients in this series had been quite free from symptoms and the tumor was accidentally discovered by

mass radiography. Another 10 cases were also diagnosed in this way although the patients had symptoms which did not cause a medical consultation. The symptoms were mostly a feeling of pressure, slight pain, and dyspnea. In 4 cases, with the tumor located at the level of the seventh cervical to the third thoracic vertebra, there was a well-developed Horner's syndrome. In 2 cases, one neurofibroma and one ganglioneuroma, the patient showed signs of von Recklinghausen's disease.

All of the patients underwent surgery.

Of the 52 patients, 49 are living three months to eleven years after the operation. Two died postoperatively (of bronchopneumonia and myocardial infarction, respectively). One died seven years after operation of cardiac disease. All the survivors, except 3, are quite free from symptoms. One patient complains of slight thoracic pains, one of slight discomfort in the hand on the side operated upon and in 1 case, the Horner syndrome has persisted. It is of interest that our 3 patients with ganglioneuroblastoma are asymptomatic 3 years, 9 months, 2 years, 3 months, and 6 months after operation, respectively. This fact has caused us to describe the ganglioneuroblastomas together with the benign tumors. In Ackerman and Taylor's[1] series of 7 cases in this group, 2 died because of metastases.

The malignant neurogenic tumors in this series are 6; i.e., 2, sympathicoblastomas, 3, sarcomas with recognizable schwannoma structures, and 1, sarcomatous mediastinal neurofibroma in a case of von Recklinghausen's disease. The sympathicoblastomas occurred in males, aged 15 and 8, respectively. The first patient was operated upon at the age of 5 for an extradural spinal tumor of suspected neuroblastoma type and ten years later presented a rapidly growing and infiltrating inoperable growth at the same level as the spinal tumor. A biopsy showed a clear sympathicoblastoma, and the boy died ten months later from metastases. The other patient had an orange-sized infiltrating tumor in the costovertebral sulcus. This patient died from cerebral metastases two months after the operation.

The three sarcomas of schwannoma type occurred in 1 man and 2 women aged 30, 31, and 51 years, respectively. The tumors were all located in the costovertebral sulcus, an orange to a cocoanut in size. It is interesting that none of them showed infiltrative growth grossly, and the surgeon reported complete removal. However, 2 of the patients died of the tumor within two years and one is living with recurrence one and one-half years after operation. One of these patients had generalized von Recklinghausen's disease.

The last patient with malignant neurogenous tumor, a woman 25 years of age, had suffered from von Recklinghausen's disease since childhood. She displayed a rapidly growing pear-shaped, seemingly well-delimited neoplasm in the costovertebral angle and had, in addition, multiple small tumors along the costal nerves. In spite of complete operation, she died with local recurrence and metastases eight months later.

CONNECTIVE TISSUE TUMORS

Fibromas are comparatively rare in the mediastinum. According to Blades,[6] 32 cases had been reported up to 1940, a number of these probably

being fibrosarcomas. In the series included in Table II there are 4 cases of fibroma. Mediastinal *myxoma* seems to be very rare.

In our series, there are 2 cases of fibroma, both located in the upper anterior mediastinum, occurring in women aged 23 and 48 years, respectively. One of these growths, though giving rise only to slight asthmatic troubles, had attained a size larger than a child's head. Both patients are alive and well four years after the operation. We have no case of mediastinal myxoma.

Fibrosarcomas are also rare in the mediastinum. Heuer and Andrus[25] (1940) collected 17 cases from the literature, which were mostly located in the posterior mediastinum. Brewer and Dolley[10] reported 4 cases of their own.

In our series, we have 5 cases, 3 in women and 2 in men, with ages ranging from 23 to 66. The size of the tumors varied between that of an orange and a large grapefruit. In 2 cases, the location was anterior; in 3, posterior-inferior, one of the latter tumors extending into the retroperitoneal region. In 2 of the cases, the tumor was found to be well encapsulated. The preoperative symptoms, in 4 of the cases (less than one year's duration) were characterized by pains, cough, oppression, and dyspnea. In only 2 cases could a complete excision be performed. One of these patients died postoperatively (pulmonary embolism), the other died from metastasis four years after the operation. Of the other 3 patients 1 died shortly after the exploratory operation. Two others are alive, 1 of them two months and the other two years postoperatively, the latter with signs of recurrence.

Tumors classed as *fibroxanthoma* are often reported as being located in the mediastinum, in locations similar to those of the neurogenic tumors. It seems probable that most of them in reality were schwannomas or neurofibromas with xanthomatous degeneration. We observed this type of degeneration in 12 of our 38 tumor cases in the schwannoma-neurofibroma group. On the other hand, we have no tumor in this series which we have found reason to class as fibroxanthoma.

Lipomas are often reported in an intrathoracic position. Thus, Keeley and associates[29] (1953) found 57 cases in the literature and added 1 case. However, 10 of them were "hour-glass" tumors, protruding subcutaneously through intercostal spaces, and 6 were "cervicomediastinal." Of the remainder, at least 10, including the case of these authors, had a mid-line, clearly mediastinal location.

In the material of Sabbatsberg hospital, there are a number of lateral or supradiaphragmatic intrathoracic lipomas. Only 1 such tumor was located in the mediastinum, this one occurred in a man, 28 years of age, and showed a double grapefruit-sized lobulated, well-encapsulated tumor in the thymic region. Microscopically, it was a lipoma of mature type with small islands of remaining normal thymic tissue dispersed between the tumor nodules. The tumor did not give any symptoms and had been discovered by mass radiography six years before the operation. The patient is well six months after the operation.

A few cases of mediastinal *liposarcoma* have been reported in the literature, the majority of them with a benign clinical course, however. We have no such specimen in our series.

Chondromas and *chondrosarcomas* with mediastinal location arise in the vertebral structures, the head of the ribs, and perhaps, also, in the sternum and costal cartilages, and grow into the posterior or anterior mediastinum. About a dozen cases have been reported in the literature. Only 1 of these seems to have been a clear chondrosarcoma (Edwards[18]).

In this series, we have 1 case of chondrosarcoma, occurring in a man, 35 years of age, with a preoperative history of only four months with cough and thoracic pains. He had a double grapefruit-sized tumor in the posterior mediastinum, probably arising in the third thoracic vertebra. Two years after incomplete surgical excision, he returned to the Clinic with an inoperable recurrence and died with one year from metastases. Histologically, we found a multinodulated, bone-infiltrating tumor tissue, mostly of a cellular, immature type, similar to embryonal cartilage, but in places of a more mature chondroma type.

MYOGENOUS TUMORS

A few cases of mediastinal *leiomyoma* have been reported in the literature. Most of them have been located posteriorly and some of them may have arisen from esophageal musculature. We have no case of leiomyoma in our series.

Rhabdomyoma is occasionally seen as a tumor of the chest wall, but no case of pure rhabdomyoma with mediastnal location is known. The case of Fox and Hospers[19] was an anterior mediastinal teratoma containing rhabdomyosarcomatous tissue. We have no such tumor or any pure rhabdomyoma in our series.

ANGIOMAS

Hemangiomas are very rarely found in the mediastinum. Seybold and coworkers[47] (1949) reported 14 cases from the literature and added 3 of their own. Of these 17 cases, 8 showed signs of malignancy. During recent years, at least 3 more cases have been described, all benign (Schlumberger,[45] Thomas and associates,[51] and Grimes and associates.[22]) Schlumberger[45] stresses the fact that mediastinal teratomas may be partially hemangiomatous. Brindley[11] (1949) reported one tumor claimed to be a mediastinal glomangioma.

The present series includes four hemangiomatous tumors and one peculiar specimen, which perhaps is a malformation rather than a tumor.

The four hemangiomatous tumors were all located in the upper anterior mediastinum; walnut to orange in size. None of the tumors were encapsulated. All showed an infiltrative growth around the upper mediastinal vessels and in 1 case infiltration into the heart and lung also. Histologically, they all presented a rather peculiar picture. Two of the tumors were of identical appearance, both benign. In the third, the microscopic picture showed malignancy, but the patient has remained well ten years after operation. In the fourth case, the tumor was an infiltratively growing hemangiopericytoma; this patient died postoperatively.

In the fifth case, a well-encapsulated walnut-sized tumor was located in the costovertebral angle and was built up by cavernous blood-filled spaces interspersed with a heavily melanin-pigmented cellular tissue.

As all of these cases are instances of rare and morphologically peculiar specimens, they will be reviewed here briefly.

CASE REPORTS

CASE 1.—A woman, aged 29, had a history of pains and a pulsating sensation in the left arm of one year's duration. A firm tumor was palpable medially above the left clavicle. At operation, a walnut-sized, firm, grayish-red tumor was found growing as a cuff around the innominate vein and the adjacent part of the subclavian vein, moderately stenosing these vessels. It was extirpated together with the infiltrated parts of the veins. The patient is quite well three and one-half years after the operation. *Histology:* The tumor tissue had a more or less abundant fibrous stroma enclosing numerous small strands of cells, rather rich in cytoplasm and forming many syncytia (Fig. 5). The cells were arranged around narrow spaces, some empty, some containing red corpuscles. There was neither nuclear polymorphism, nor mitoses.

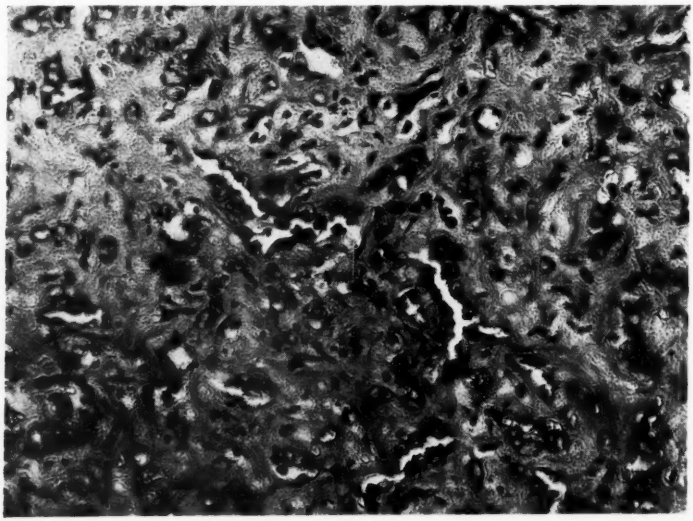
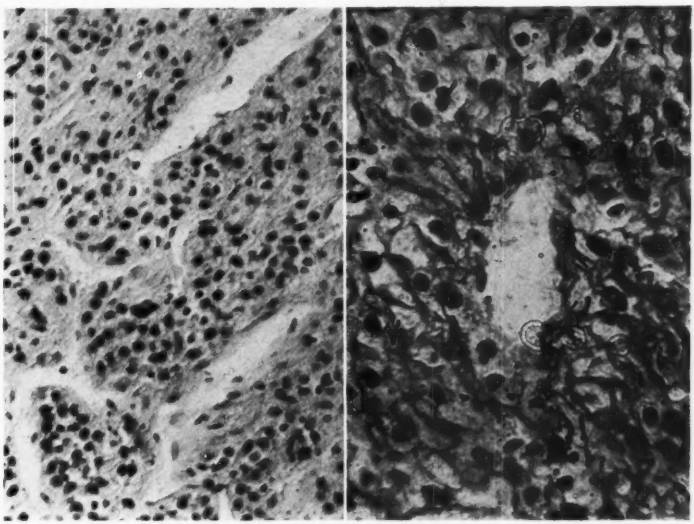


Fig. 5.—Case 1. Capillary hemangioma with syncytial endothelium cells. (van Gieson stain, ×430.)

CASE 2.—A man, aged 76, who had a history of uncertain duration of oppression, pains, dyspnea, swallowing troubles, and hoarseness. X-ray showed an upper mediastinal tumor with dislocation of the trachea and esophagus to the right. At thoracotomy a firm, orange-sized neoplasm was found in the upper anterior mediastinum, free from the lung, but obviously infiltrating around the big vessels. A frozen-section biopsy was erroneously interpreted as cancer and no attempt at surgical removal was made. After subsequent x-ray therapy, he was subjectively improved three months postoperatively. *Histology:* The same as in Case 1.

CASE 3.—A woman, aged 32, had a history of fatigue, raised temperature, cough, and tachycardia of 20 months' duration. Thoracotomy revealed an upper mediastinal neoplasm of grapefruit size, growing around the aorta ascendens and infiltrating through the pericardium, the wall of the left auricle, and the region of the left coronary sulcus (Fig. 6). The neoplasm was adherent to the lung and infiltrated the left upper lobe in one place. It was solid, grayish red, vascular, with necrotic patches. The patient died, postoperatively, after a partial removal. *Histology:* It was a cellular tumor with abundant narrow blood spaces,

Fig. 6.—Case 3. Roentgenogram of a mediastinal hemangiopericytoma.



A. *B.*

Fig. 7.—Case 3. Hemangiopericytoma. (*A*, van Gieson, ×230; *B*, silver impregnation according to Laidlaw, ×560.)

Fig. 8.—Case 4. Sarcomatous hemangioma. Strands of syncytial tumor cells imitating capillaries. (van Gieson stain, ×280.)

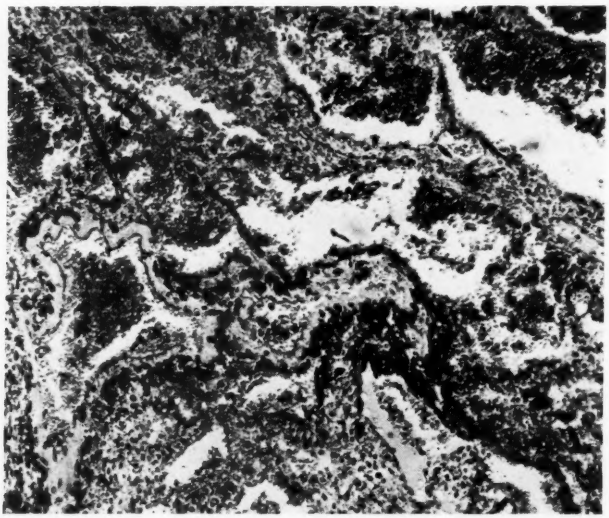


Fig. 9.—Case 5. Melanin-pigmented cavernoma. (van Gieson stain, ×140.)

lined with endothelial cells (Fig. 7). Peripheral to the lining cells were numerous polyhedral cells, rather rich in cytoplasm and with a moderate nuclear pleomorphism. There was a well-developed intracellular reticulum with a clear arrangement around the capillary spaces, which corresponded to the tumor called hemangiopericytoma, described by Stout[50] (1949).

CASE 4.—A man, aged 41, had an upper mediastinal tumor discovered by x-ray in connection with an acute respiratory infection. It grew slowly and symptomless during one year, giving slight thoracic pains only 6 weeks before operation. Thoracotomy revealed an orange-sized upper anterior mediastinal tumor growing infiltratively around the aortic arch and other big vessels and also infiltrating the pericardium. The cut surface was grayish with necrotic spots. The surgeon was able to remove the neoplasm in radical fashion. The patient is alive and well more than ten years after the operation. *Histology:* In the central parts, a clearly sarcomatous cellular tissue with bundles of elongated pleomorphic, mitosis-containing cells surrounding numerous small capillary spaces was seen. In the periphery, the tumor cells grew in long, often bifurcated strands in a loose connective tissue. The strands were built up by two layers of syncytial cells enclosing narrow spaces with a few red corpuscles, thus imitating young, newly formed capillaries (Fig. 8). It was classed as hemangiosarcoma.

CASE 5.—A woman, aged 55, had had coughing spells for 15 years. Six years before the operation, a mediastinal tumor was discovered by x-ray. Tests did not reveal any increase in size, and no new symptoms appeared. Thoracotomy revealed a walnut-sized well-encapsulated tumor in the right costovertebral sulcus at the level of the fourth thoracic vertebra. It seemed to be connected with the intercostal nerve, but had no intraforaminal extension. It was bluish in color with multiple blood-filled cystic spaces. After radical extirpation, the patient has remained well for more than two years. *Histology*: The tissue contained numerous cavernous blood-filled spaces with a thin endothelial covering. Between the spaces, there was a cellular tissue with small monomorphous, round nuclei and indistinct cell borders. The cytoplasm contained abundant fine or coarse granules of darkish-brown pigment, which presented a positive reaction to Masson's melanin stain. There was also much iron-positive pigment in places, probably hemosiderin. The cellular tissue revealed no structures similar to Schwannoma or neurofibroma and no nerve cells were to be found. We were not able to classify this specimen or to find any of similar description in the literature. We believe that it may possibly be a hamartoma of combined vascular and neurogenic origin (Fig. 9.).

Lymphangiomas in the mediastinum seem to be slightly more common than hemangiomas. According to Schlumberger,[44] 12 cases had been recorded up to 1948. In the series of Brewer and Dolley[10] (1949), Curreri and Gale[14] (1949), and Conklin[13] (1950), there are 3 more cases and Lob[36] has published 1 case (1951). Thus, at least 16 cases are known. The tumors have occurred predominantly in adults in an anterior location and have been mostly without symptoms.

In this series, we have 7 cases of lymphangioma (3 in men and 4 in women). Two of them were children (3, and 6 years of age); the others adults (whose ages ranged between 36 and 48 years). In our series, the anterior location does not predominate. We had 2 situated upper-anteriorly, 1 anteriorly in the hilar region, and 1 on the left side of the pericardial sac. On the other hand, we have 3 cases with lower posterior location, the tumor growing paravertebrally and supradiaphragmatically behind the heart, adherent to the pericardium. The smallest specimens have been about the size of an orange, the largest one covered the left side of the pericardium and had an extension of about 20 by 30 cm, but was not very thick. All the tumors

were multicystic, either with a honeycombed cut surface throughout or with one main cyst. In 2 cases there were small extra cysts, separated from the main tumor. The cysts contained a water-clear or yellow-brownish fluid. Microscopically, the picture was characteristic: cystic spaces with an endothelial covering and thin fibrous wall, and in every case the specimen contained scattered streaks of smooth musculature. One may also find sparse lymphocyte infiltrates. The stroma tissue between the cysts always contained adipose tissue. In the 2 cases with upper anterior location, we did not find any thymus tissue in the stroma.

There is always some difficulty in differentiating mediastinal lymphangioma from thymic cyst and pericardial cyst. In our opinion, a thymic cyst seldom has smooth musculature in the wall and, if one had adequate material, one will always find rests of thymus tissue with Hassall's corpuscles adjacent to the cyst wall. The pericardial cysts do not show any smooth musculature, and the lining cells are (at least sometimes) cuboidal and not of the flat, endothelial type.

These lymphangiomas grow slowly and are symptomless. In all cases but one in our series, the tumor was discovered accidentally, either by mass radiography or by x-ray examination in connection with an acute respiratory infection. In the remaining case, there had been thoracic pains, dyspnea, and attacks of suffocation. In all the cases, it was possible to remove the tumor radically and all the patients are alive and well one and one-half to ten years after the operation.

LYMPHOSARCOMA AND PLASMACYTOSARCOMA

Malignant lymphoma, including Hodgkin's disease and leukemia, often produces mediastinal masses, especially in the upper anterior part of the mediastinum. Thus, patients are often operated upon suspected of having mediastinal tumors. In quite a few such cases, an erroneous histologic diagnosis of malignant thymoma is given. We have seen many instances of this in the material from this Clinic.

The lymphosarcomas do not constitute a very large group among the mediastinal neoplasms. In the series recorded in Table II, they averaged less than 3 per cent of all mediastinal tumors and cysts.

Plasmacytoma and plasmacytosarcoma seem to be extremely rare in the mediastinum. We have found only 1 case of the former (Childress and Adie[12]), and 1 of the latter (Divis[16]) reported in the literature.

In the present series, we have (with exclusion of Hodgkin's disease and leukemia) taken up 5 cases of mediastinal lymphosarcoma and reticulum-cell sarcoma, and 1 case of plasmacytosarcoma.

The 5 cases of lympho- or reticulum-cell sarcoma occurred in 3 women and 2 men, whose ages ranged from 10 to 80 years. The preoperative course was mostly less than 6 months, only in one case being as long as three years. The symptoms were most often oppression dyspnea, cough, thoracic pains, fatigue, loss of weight, and sometimes swallowing difficulty. All tumors were located in the upper anterior region varying in size between an orange and a fist, and all were growing infiltratively. The tumor masses were soft,

grayish-white, sometimes nodulated, sometimes with necrosis and small cysts. Histologically, only 1 of the cases was a pure lymphocytosarcoma, the others were reticulum-cell sarcomas of small, large celled or polymorphocellular type. In 2 of the cases, the surgeon tried to remove the tumor radically, in 2, partial resection was performed. The fifth patient was never operated upon. The autopsy in this case did not reveal any dissemination of the tumor. All of the patients operated upon, in spite of radiologic treatment, have died within two and one-half years after the operation.

The case of plasmacytosarcoma occurred in a man of 37, presenting a two month's history of pains in the left arm and shoulder. The thoracotomy revealed a tumor, the size of a hen's egg, in the upper posterior mediastinum, surrounding the three uppermost thoracic vertebras, growing into the intervertebral foramina and surrounding the intercostal nerves and the left brachial nerve plexus. X-ray exploration did not reveal any involvement of the thoracic vertebras, nor any signs of myeloma in other parts of the bone system. No albumin was found in the urine. Histologically, the tumor tissue was built up by pleomorphic, mitose-containing but mostly well-recognizable plasma cells. The tumor was considered inoperable and only a biopsy was taken. The patient died of the tumor one and one-half years after the operation.

THYMOMAS

There is much controversy concerning the definition of the term thymoma. Many authors have described malignant widely metastasing tumors, supposedly originating in the upper anterior mediastinum, as thymic carcinoma, and the term thymic lymphosarcoma has also been introduced. Without a doubt, the majority of the so-called thymic carcinomas have been either pulmonary carcinomas with mediastinal extension or malignant teratomas with one-sided development of the epithelial component. We, also, believe that upper mediastinal lymphosarcomas originate from lymph nodes in this region and not from the thymic tissue. Thus, we entirely agree with the definition recently given by Seybold and associates,[47] "thymoma is a slowly growing tumor of the thymus which has arisen from both the epithelial and lymphocytic elements of the thymic parenchyma." A thymoma may grow infiltratively in adjacent structures and give implantation metastasis in the pleura or pericardium. Clearly documentated cases of thymoma, as previously defined, with extrathoracic metastasis, are—if existent—extremely rare.

It has been suggested that those thymomas which are associated with myasthenia gravis fall into a distinct group. In all cases of myasthenia gravis, the tumors seem to have fulfilled the criteria given previously, and no case with extrathoracic metastasis is clearly documented. However, most thymomas with these morphologic and biologic properties are not associated with myasthenia gravis, and in our opinion it is impossible to find any histologic difference between the two groups.

Thymoma constitutes a rather large proportion of the mediastinal tumors and cysts. In the previous series, presented in Table II, it ranks fifth, in our series cases of thymoma come third after those of neurogenic tumors

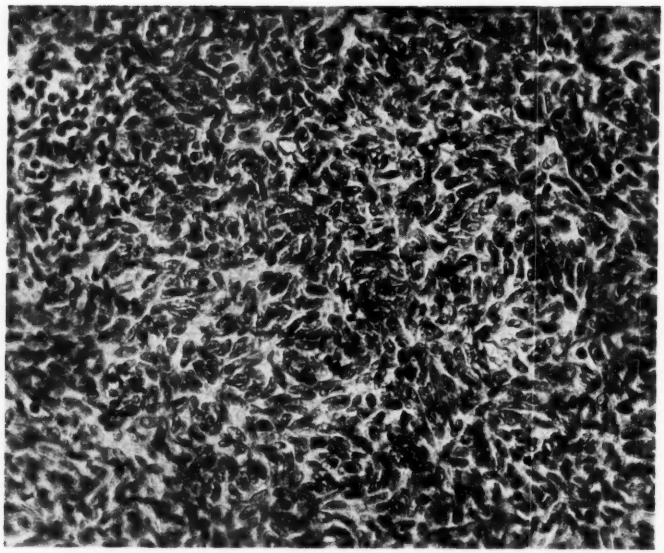


Fig. 10.—Thymoma of epithelial type. Note slight admixture of lymphocytes in the upper left corner. (van Gieson stain, ×280.)

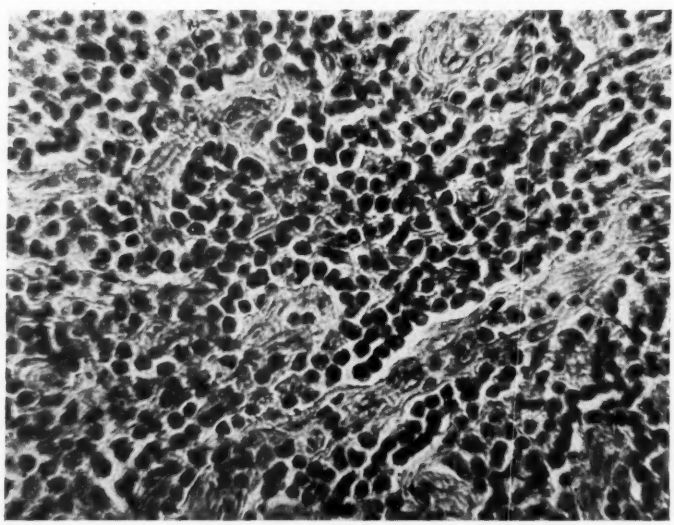


Fig. 11.—Thymoma of lymphocytic type with a perithelial arrangement of the thymo-epithelial cells (so-called reticular perithelioma of the thymus according to Pope and Osgood). (van Gieson stain, ×560.)

and bronchial cysts, which makes it more common than teratomas. Because of the confusion as to the definition of thymoma, it is impossible to give even an approximate evaluation of the number of cases of thymoma described or referred to in the literature.

This series includes 19 cases of thymoma. The sex distribution is even (9 men and 10 women). The ages range between 20 and 67 years. The mean age for both sexes is 47 years, but is 42 for men and 52 for women.

Thymomas are predominantly located in the upper anterior mediastinum, but location in the pulmonary hilar region is not rare, and even thymomas located on the neck and in the anterior inferior mediastinum have been described in the literature (Alter and Onato,[4] Ringertz,[41] Hardymoon,[24] and others). In this series, the site was the thymic region in 16 cases and the pulmonary hilar region in 3 cases.

The size varied between that of a mandarin orange and a large grapefruit (12 by 10 by 8 cm.). The tumors were rounded or flattened, often shaped as the thymus. The gross picture was characteristic in all cases: a nodulated tumor with broad streaks of fibrous stroma surrounding islands of soft grayish-white tumor tissue. The latter may show cystic degeneration (in about one-third of our cases) and cholesterol and lime salts may be deposited in the stroma. Infiltrative growth into adjacent structures was reported by the surgeon in only 1 case, and we were not able to establish this phenomenon, microscopically, in any other case. In this aspect, our series differs from that of Seybold and co-workers,[48] who found infiltrative growth in one-fourth of their cases. In the previously mentioned case of invasive tumor, there were also pericardial implantation metastases, and in one other case, multiple pleural implants.

Microscopically, all the tumors in our series presented principally the same picture; the growth being made up of lymphocytes and thymo-epithelial cells. However, the proportion between the two kinds of cells varied very much, not only in the different tumors, but also in different parts of the same neoplasm. In 5 cases, the lymphocytes were definitely dominating, 5 showed about equal distribution, and 9 were predominantly epithelial. In the latter group, areas of purely epithelial tissue occurred (Fig. 10), but we always found other parts with lymphocytic admixture. In the epithelial areas, the fusiform cells showed a tendency to form knots and whorls, often with a pericapillar arrangement. We also found this whorl-like pericapillar proliferation of thymo-epithelial cells in an otherwise very lymphocyte-rich thymoma (Fig. 11—This picture probably corresponds to Case 6 in Pope and Osgood's[39] series of "reticular perithelioma" of the thymus). Hassall's corpuscles, more or less well developed, were found in 5 (nearly 25 per cent) of our cases, which tallies well with the frequency in the series of Seybold and associates.[48] A malignant cell picture was present in 2 cases which were not identical with those showing invasive growth and metastases. Thus, some sign of malignancy was found in 4 out of 19 cases.

The preoperative course was, in 5 of our cases, domiated by more or less pronounced symptoms of myasthenia gravis, in 1 case for twelve years. In 3 cases without this disease, there were only slight local symptoms such as pains and dyspnea. In 1 case, the thymoma was accidentally discovered during the course of agranulocytosis. In the remaining 10 cases, the patients were asymptomatic and the tumor was discovered by mass radiography.

Thus, we have 5 cases with preoperative myasthenia gravis. In another case, this disease developed after the operation and was obviously associated with the outgrowth of pleural metastases. These were removed, but the patient died four years after the first operation from myasthenia gravis. The autopsy revealed a local recurrence also. This makes, in all, 6 myasthenia gravis cases among 19 cases of thymoma; i.e., 32 per cent, a much lower figure than in the series of Seybold and co-workers[48] (75 per cent). The frequency of thymoma in myasthenia gravis is at least 15 per cent (Good[21]), in a Swedish series (Ringertz[41]) the percentage was 33. Four of the 6 patients with myasthenia gravis in our material were women with an average age of 49 while the two men were aged 20 and 21.

In all of our 19 cases, a mediastinal tumor was recognized roentgenologically.

Two of the patients were never operated upon, in 1 case because the woman died of myasthenia gravis before the planned operation could be performed, and in 1 case, the patient died of agranulocytosis. In 1 of the 17 operated cases, a biopsy was performed without further surgery (due to an erroneous first diagnosis of lymphosarcoma), and in 1 myasthenia gravis case with pericardial infiltration and metastases, a radical removal was impossible. In the remaining 15 cases, the tumor was radically extirpated.

The postoperative course in 11 of the 12 patients without myasthenia gravis, who were operated upon was without remark, and 10 of them are living free from symptoms one-half to four and one-half years after the operation. One patient has died of unknown cause and 1 by a traffic accident. Of the 5 patients operated upon with myasthenia gravis, 1 died postoperatively in respiratory insufficiency and 1 succumbed with myasthenia gravis due to metastases (see above). Of the remaining 3, 1 is quite symptom-free two and one-half years after the operation, 1 moderately improved after three years, and 1 slightly improved after one-half year.

THYMIC CYSTS

In the early literature, "thymic cysts" were frequently reported in syphilitic patients, especially in children with congenital syphilis. Nowadays, such lesions are rarely reported. On the other hand, a number of cysts claimed to have arisen from remnants of the thymic duct have been described in recent years. Krech and associates[31] (1954) collected 13 such cases from the literature and added 2 of their own. In a number of these collected cases, the cysts had a lining of high cylindrical, even ciliated epithelium, stratified or squamous epithelium. Many kinds of cysts may be located within the thymic region; teratomas of epidermoid cyst type, bronchial cysts, and

cystic lymphangiomas. In our opinion, one must be very careful with the differential diagnosis. One cyst in this series we have classed as thymic cyst because of the widely distributed layer of thymic tissue with Hassall's corpuscles surrounding the thin fibrous, endothelium-lined wall. Due to the fact, however, that sparse bundles of smooth musculature were present in the wall, we suspect that this cyst might be an intrathymic lymphangioma. It occurred in a man of 61 years of age with two years' history of oppression and dyspnea. The cyst was of the size of a double fist, stalked from the thymic region and protruded into the right pleural cavity. The patient died five years postoperatively of gastric carcinoma. No autopsy was performed.

TERATOMAS

The mediastinum is, after the gonads, the most common site of teratoma in the human body. These neoplasms constitute a considerable proportion of the mediastinal tumors and cysts. Rusby,[43] in 1944, found 251 cases in the literature, and we have seen at least 100 more cases referred to after that year. The average percentage of teratomas in the series included in Table II is about 21, the teratomas ranking second after the neurogenic tumors. In our material, however, they rank fourth after the neurogenic group, bronchial cysts, and thymomas, with 10.3 per cent.

The frequency of malignancy among the mediastinal teratomas was 13 per cent in Rusby's collected material. Other literature compilations (Brewer and co-workers,[10] Laipply[32]) have shown 12.9 and 11.4 per cent, respectively. In some recent consecutive series, a higher percentage is shown, e.g., Hanner and associates[23] (1952), 33.3 per cent.

The sex distribution is even in the benign group, but it is pointed out that malignant teratomas predominantly occur in men (Fralic and Welsman[20], 1951, 8 males to 1 female). Most mediastinal teratomas occur in young adults. According to Willis,[55] the average age is 27 years. Laipply[32] states that 70 per cent of the patients are younger than 31 years and Rusby[43] that in 80 per cent of the patients the age was between 10 and 40 years.

The majority of recent authors consider the genesis of mediastinal teratoma to be associated with the development of the thymus (Schlumberger,[44] 1946). It is probable that all benign teratomas are tridermomas. However, in most cases, the ectodermal structures dominate so strongly that only a comprehensive histologic examination reveals the other structures. Because of this fact, and their common gross appearance of a cyst containing hairs and sebaceous matter, they have often been called dermoid cysts. The relatively high frequency of endodermal structures and, above all, of pancreatic tissue in the mediastinal teratomas has been pointed out in the literature. It is also stated that the malignant teratomas in this region lack ectodermal structures and that their epithelial component is predominantly adenocarcinomatous. The mesodermal structures are mainly low differentiated, though occasionally islets of cartilage and cysts of endodermal type may occur. Rare cases with a predominance of choriocarcinomatous tissue have been described in males (Laipply and Shipley,[33] 1945).

Our series contains 14 benign and 2 malignant teratomas, which gives a malignancy percentage of 12.5, which agrees with the figures of Rusby,[43] Brewer and Dolley,[10] and Laipply[32].

The *benign teratomas* showed an even sex distribution (7 males, 7 females), and an average age of 30. The location was the thymic region in 10 cases, the pulmonary hilus region in 3, while the site in 1 case was the costovertebral sulcus at the level of the third to the seventh thoracic vertebra. (This posterior location is rare, Rusby[43] reports only 3 such cases out of 251.) The size varied between duck's egg and melon, one-half of the specimens being at least the size of a grapefruit. All the benign tumors were well delineated and all but 1 had the gross appearance of a cyst with more or less pronounced mural nodule or nodules. The content was hairs and sebaceous matter in 10 cases, detritus only in 3. In no case were any teeth observed. One tumor in the thymic region was polycystic with solid tissue between the cysts. In 1 case, there was a fistula between the cyst and the main bronchus; this patient had expectorated hairs and showed multiple pulmonary abscesses.

Microscopically, 11 of these 14 tumors exhibited tissues from the three germinal layers in spite of the fact that the examination material was limited in several cases. In one well-examined case, only meso- and endodermal structures were found. As the endodermal structures in this cystic tumor did not appear as well-organized gastrointestinal mucosa, we have not classed the case as gastroenteric cyst. In 2 cases, endodermal structures were lacking, but in these cases the material was limited. The most common structures were epidermal with hair follicles, sweat and sebaceous glands; but cysts lined with respiratory epithelium, and with smooth musculature and mucous glands in the wall, were just as common. Cysts lined with epithelium of enteric type occurred in 7 cases, well-developed pancreas tissue in 3. Glia tissue was found only in 5 cases (36 per cent). Willis[55] states that its frequency in all teratomas is at least 80 per cent. We were not able definitely to identify nerve cells in any case. Cartilage and bone were present in 6 cases. Striated musculature and pigmented epithelium were found, each in 1 case.

Normal thymus tissue was found adjacent to the capsule in 6 of the cases, including 2 with pulmonary hilar position. No doubt this phenomenon would have been noted more frequently if we had been able to examine all the tumors in toto.

In 4 cases there were no symptoms, the remaining patients had a history of thoracic pains, dyspnea, and cough. In the bronchial fistula case, the patient had recurrent pneumonia and had expectorated hairs. Most of these tumors are obviously slow growing, 5 tumors—less than the size of a fist at operation—had been discovered three to eight years previously.

All the tumors were radically removed. One patient could not be traced afterward. All the others are living free from symptoms, 5 months to 10 years after the operation.

Our 2 cases of *malignant teratoma* occurred in men, aged 30 and 36, respectively. In both cases, the tumor was located in the thymic region,

being about grapefruit sized, and showing a pronounced infiltrative growth. In 1 case, where the neoplasm could be nonradically removed, its tissue was predominantly solid, grayish white and soft in consistency.

Microscopically, 1 of the cases presented an entirely dominating epithelial component of highly malignant aspect, growing in festoons and adenomatous structures. The sparse connective tissue was loose, myxomalike, without any cartilage or bone differentiation. However, 1 small cyst with highly differentiated squamous epithelium was found. The other tumor also presented, besides a low-differentiated malignant epithelial proliferation, more differentiated endo- and mesodermal structures, such as cysts with ciliated columnar epithelium, smooth musculature, and mature connective tissue.

Both patients had a preoperative history of less than three months of pains, fever, dyspnea, and hemorrhagic cough. In 1 case, the tumor was not radically removed, in the other, only a biopsy was performed. Both patients died within six months after the operation.

MEDIASTINAL "BRONCHIAL ADENOMAS"

We have in this series two specimens of mediastinal tumor which were morphologically and clinically benign. Their histologic picture was, at least in most parts of the tissue, very similar to that of bronchial adenoma. These tumors were well encapsulated, large as a cocoanut, and the surgeon did not find any connection between the tumors and the bronchial tree. We have not been able to find any identical cases described in the literature.

Our material contains 1 more case of epithelial tumor, definitely primary, in the upper anterior mediastinum, but with a clearly malignant histologic picture. The structure was, in this case, in principle similar to that in certain parts of one of the benign tumors mentioned before. On the other hand, the epithelial component, growing in festoons and alveolar structures, presented a picture very similar to this component of a malignant teratoma. As we were unable to establish organoid features in any part of this tumor, we have not classed it as a malignant teratoma but prefer to look upon it as a histologically malignant variant of the first-mentioned tumors.

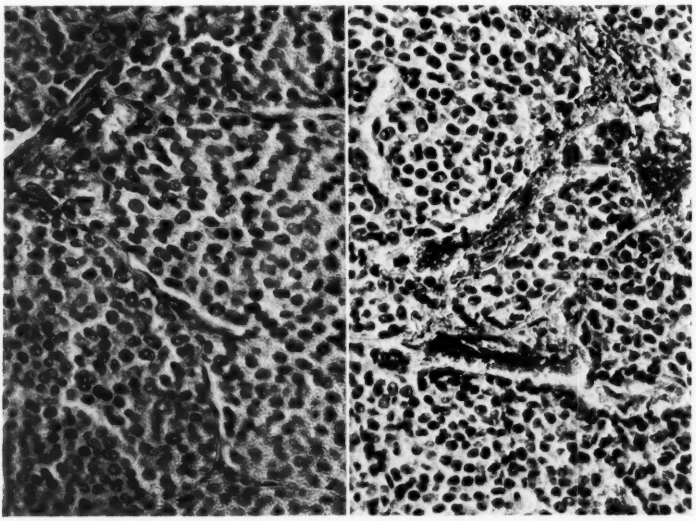
We give here a short review of the findings in these 3 cases.

CASE REPORTS

CASE 1.—A man, aged 64 had a six months' preoperative history of dyspnea and severe cough. Roentgenologically, a large upper anterior mediastinal tumor was shown. The thoracotomy revealed a well-encapsulated growth, large as a large grapefruit, adherent to the right lung, dislocating the heart and the big vessels to the left. The tissue was soft, solid, yellowish-white with some necrotic patches. The tumor was radically removed and the patient is free from symptoms five and one-half years after operation. *Histology*: In some parts the tumor tissue was built up by monomorphous polyhedral epithelial cells with small round nuclei. The cells were arranged in rounded clusters encased by narrow streaks of vascular stroma. This picture was quite similar to that of bronchial adenoma of the carcinoid type (Fig. 12, *A*). In other parts, the epithelial clusters merge into extensive solid masses penetrated by stroma streaks (Fig. 12, *B*). No mitoses or any infiltrative growth in the capsular region were found.

CASE 2.—A woman, aged 43, had no preoperative symptoms. The tumor was discovered by mass radiography six months before operation. The tumor was located in the lower

posterior mediastinum and bulged into the left pleural cavity behind the heart. It was well encapsulated with a coarsly nodulated surface. The interior tissue showed cystic degeneration leaving papillary nodules of tumor tissue next to the capsule. The growth was radically removed and the patient was free from symptoms six years after operation. *Histology*: In some areas, this tumor presented the same picture as in the first case (see Fig. 12, *B*, and Fig. 13, *A*) but in other parts there was a peculiar structure with elongated cells showing a palisaded or rosette like nuclear arrangement (Fig. 13, *B*).

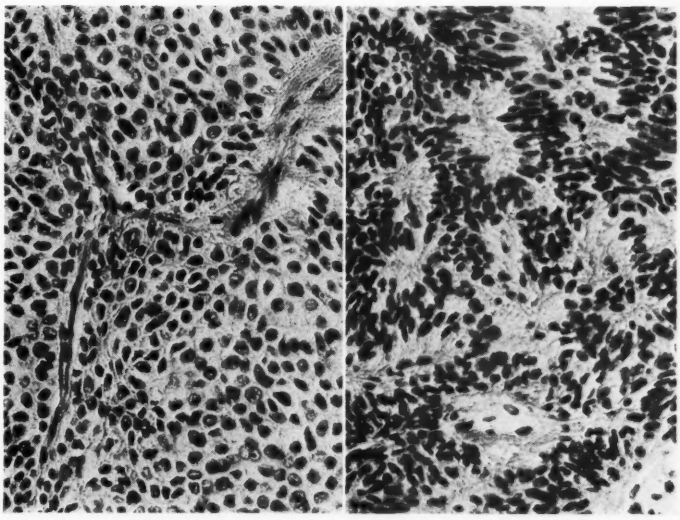


A. *B.*

Fig. 12.—Case 1. Bronchial adenoma of the mediastinum. *A*, Solid clusters of tumor cells (carcinoid type); *B*, another part of the same tumor: diffuse cellular arrangement and sparse narrow stroma streaks. (van Gieson stain, ×280.)

Case 3.—A man, aged 39, after six months' symptoms of progressive dyspnea and arm edema, had a large upper anterior mediastinal tumor discovered by x-ray. A puncture biopsy showed a malignant, predominantly epithelial tumor, possibly of the teratoma type. The patient was treated radiologically for two years and ten months. X-ray revealed a slow progression of the growth, and due to the progressive dyspnea, it was decided to operate. At thoracotomy, more than three years after the first symptoms, an infiltratively growing retrosternal tumor was found. It was as large as a cocoanut, and bulged into the right pleural cavity and invaded around the big vessels and into the pulmonary hilar region. The patient died postoperatively and the autopsy showed remaining tumor tissue in the hilar region but no metastasis. *Histology*: The solid tumor had a uniform structure throughout. It was built up by epithelial cells growing in festoons and alveolar formations in a scanty loose nonmyxomatous stroma. There was an evident cellular polymorphism and many mitoses (Fig. 14). The arrangement of the epithelial cells was rather similar to that in Case 2 (see Fig. 13, *B*).

The pathogenesis of these tumors is difficult to interpret. They cannot possibly be exclusively centripetally growing bronchial adenomas, as none of them showed any anatomic connection with the bronchial tree. In Case 2, the gross aspect of the tumor might suggest the formation of an adenoma



A. *B.*

Fig. 13.—Case 2. Bronchial adenoma of the mediastinum. *A*, Same type of tumor tissue as in Fig. 12, *B*. *B*, Another part of the same tumor with a rhythmic nuclear arrangement and formation of rosettes. (van Gieson stain, ×280.)

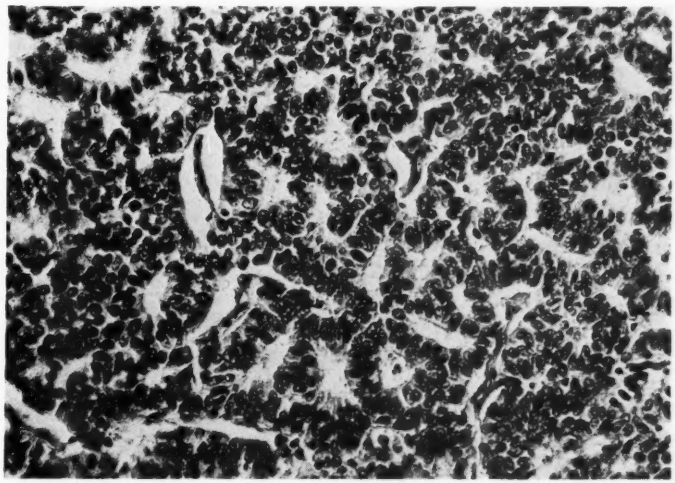


Fig. 14.—Malignant mediastinal epithelioma, probably belonging to the bronchial adenoma group. Note a certain similarity in the cellular arrangement to that in Fig. 13, *B*. (van Gieson stain, ×280.)

in a bronchial cyst, but histologic examination did not reveal any rest of bronchial cyst wall in the periphery. Possibly these tumors are teratomas with a one-sided differentiation, in which only a tissue, derived from the embryonal bronchial anlage, has been developed. Such an interpretation would explain the fact that the picture in Case 3 is so very similar to the epithelial component in malignant mediastinal teratoma.

BRONCHIAL CYSTS

These cysts are rather common mediastinal growths. Dodds[17] collected 85 cases in the literature up to 1950, and after that time at least 10 cases have been published. In the combined series recorded in Table II, they rank third after neurogenic tumors and teratomas. They occur characteristically in the posterior mediastinum at the level of the tracheal bifurcation and pulmonary hilum. On the basis of their location, Maier[37] has classified them into four groups; paratracheal, carinal, hilar, and paraesophageal.

The origin of bronchial cysts has been attributed to displacement of embryonal tissue. They are thought to arise from an additional budding of the foregut, equivalent to the respiratory anlage, which is carried down into the mediastinum. Thus, their development should relate them to accessory lobes of the lung.

Only a few of the cases reported in the literature have shown bronchial communication (e.g., one of Allison's[3] cases). Laipply[32] is of the opinion that such cases are bronchial diverticula and not real malformation cysts.

In our series, we have classed 21 specimens in this group. Nineteen of them presented the classical picture of bronchial cyst whereas 2 were multicystic formations with a structure resembling a malformed embryonal lung. We have not been able to find any cases in the literature similar to this latter group.

The 19 first mentioned cases occurred in 12 men and 7 women, whose ages ranged between 16 and 52, with an average age of 37 years. All but 4 were located in the posterior mediastinum. Eight cysts were located above, 5 below the level of the pulmonary hilum, and 6 in this level. As many of the cysts were fairly large, we found it difficult to group all of our cysts according to Maier's system. However, one was definitely paratracheal, three carinal, and six hilar. Their size varied between that of a hen's egg and a melon, 8 being at least grapefruit sized. Grossly, they were unilocular, thin-walled, the content being either clear, mucinous or brownish, or detrituslike (often mistaken by the surgeons for dermoid cysts). In 1 case, the cyst presented a narrow communication with the bronchial tree. We do not consider this goose egg sized cyst to be a bronchial diverticulum, especially as irregular patches of cartilage occurred in its wall.

The microscopic picture is characteristic, showing a lining of well-developed respiratory epithelium, often, in small areas, showing metaplasia toward squamous type. The thin fibrous wall always contains streaks of smooth musculature and mostly mucous glands. In 5 of our cases, irregular patches of adult cartilage were found in the wall. In 3 cases, we found small accessory lumina with respiratory epithelium included in the wall.

In 8 of our 19 cases (42 per cent), the cyst had not caused any symptoms and had been discovered by mass radiography (more than five years before the operation in 6 of these 8 cases). This percentage tallies well with Hirschfeld's statement that 60 per cent of the bronchial cysts give symptoms. In the other 11 cases, there was a preoperative course of up to six years with slight troubles, mostly cough, dyspnea, and oppression. All the patients had radical operations performed and are alive and well, ten months to ten years after the operation.

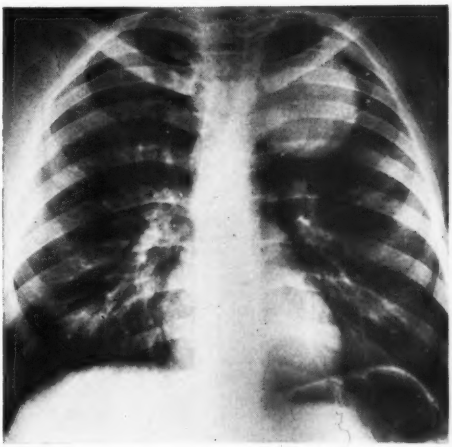
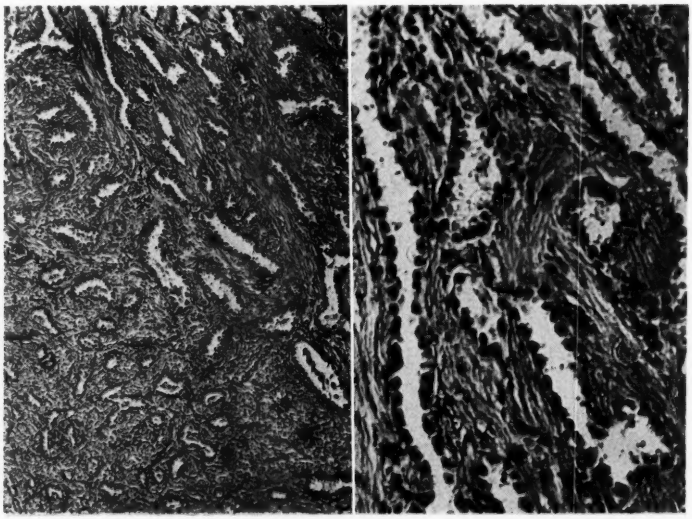


Fig. 15.—Roentgenogram of a malformation tumor of the "lung anlage" type. (See Fig. 17.)

The 2 multicystic specimens, mentioned previously, are very interesting, because they confirm the occurrence in this region of cysts with a structure resembling that of an embryonal lung. This is in consonance with the theory that bronchial cysts develop from an accessory pulmonary anlage. They occurred in one man and one woman, aged 20 and 32, respectively. They had both the same location, superiorly in the costovertebral sulcus, one plum and one grapefruit sized (Fig. 15). They were separated from the lung and showed no communication with the bronchial tree. Grossly, these formations were well encapsulated, the cut surface having a honeycombed appearance with multiple, small cysts. Microscopically, the 2 specimens seemed to reproduce different stages of the embryonal pulmonary development. Thus, in the small one the stroma tissue was built up by fibrous tissue and smooth musculature (Fig. 16). Encased in this tissue were multiple, small, elongated, and often bifurcated lumina lined with rather large, cuboidal, or low cylindrical nonciliated cells, resembling the cells in the early embryonal bronchial buds. No mucous glands or cartilage was present. The other specimen represented a more mature structure (Fig. 17). Here the cystic spaces were larger and covered by a mature respiratory epithelium. The fibrous stroma contained abundant streaks of smooth musculature often concentrically arranged around the cystic spaces. In this case, there was no admixture of cartilage or mucous glands, although the epithelium often contained mucous vacuoles.

In both these cases, the tumors grew slowly and without symptoms, and were discovered by mass radiography (in 1 of the cases, 9 years before the operation). They were easy to remove radically. The woman is alive and well five years after the operation. The man, however, (with the specimen



A. *B.*

Fig. 16.—Malformation tumor of immature "lung anlage" type. *A*, van Gieson stain, ×55. *B*, Detail of the upper left corner. (van Gieson stain, ×230.)

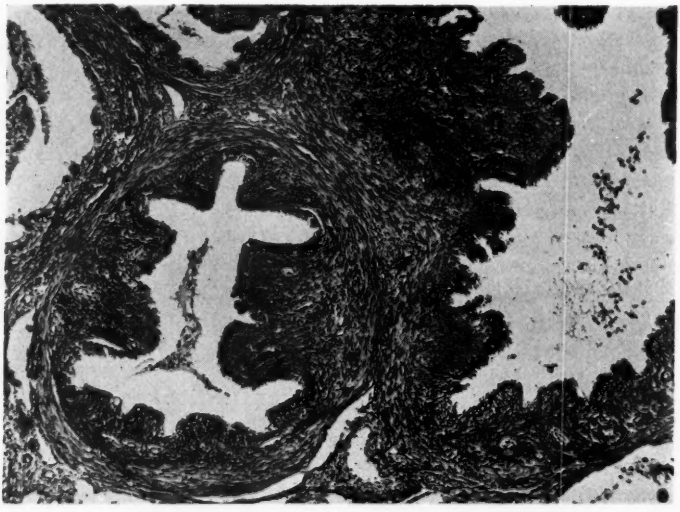


Fig. 17.—Malformation tumor of mature "lung anlage" type. Cystic spaces with cylindrical epithelium and concentrically arranged streaks of smooth musculature. (van Gieson stain, ×55; same magnification as in Fig. 16, *A*.)

of more adult type) developed a rapidly growing testicular tumor five months after the operation which was extirpated and showed the typical picture of seminoma. He died with signs of metastases seven months later. Unfortunately, no autopsy was performed. We feel strongly that the mediastinal affection with its definitively benign structure was in no way related to the malignant testicular tumor.

PERICARDIAL CYSTS

A fairly large number of thin-walled cysts with endothelial lining and a location close to the pericardium have been classed under this heading in the literature. Thus, Hirschfeld,[26] in 1951, was able to collect 72 such cases. Occasionally, these cysts show communication with the pericardial sac. In such cases, it is almost impossible to differentiate between pericardial diverticulum and real malformation cyst, due to deficient fusing of the primitive celomic lacunae. Moreover, some authors (e.g., Lambert,[34] and Leahy and Calver[35]) consider pericardial cysts and diverticula to be the same thing.

It may also be difficult to differentiate between pericardial cyst and lymphagioma. Grossly, these two growths may be very similar. However, in our opinion, it is possible to separate them microscopically, because the lymphangiomas nearly always have streaks of smooth musculature and small lymphocyte infiltrates in the walls. We also feel that the lining cells in pericardial cysts, at least in patches, are cuboidal rather than flat endothelial. In the combined materials, presented in Table II there are 1.6 per cent lymphangiomas and 10.4 per cent pericardial cysts, whereas in our series the figures are 4.5 per cent and 3.9 per cent, respectively. We suspect that some of the pericardial cysts frequently reported in the literature really are lymphangiomas.

Our series contains 6 cases, 4 men and 2 women with ages ranging between 31 and 56, in average 41 years (Hirschfeld[26] found no case in the literature younger than 27). The cysts were all located in the anterior inferior mediastinum, in 4 cases on the right and in 2 on the left side of the pericardial sac. The largest cyst, right-sided, measured about 24 by 20 by 20 cm., the smallest one was the size of a hen's egg. In one case, the cyst showed a 1 cm. wide pericardial communication. In another case, there was a small cyst which stalked from the main one. All the cysts contained a clear nonviscous fluid and had a thin, purely fibrous wall with a lining of single row of cuboidal or flat cells.

In 5 of our 6 cases, there were no preoperative symptoms, the cysts being discovered by mass radiography (Hirschfeld[26] reports a symptom-free preoperative course in 39 of his 72 literature cases). Our remaining case had a six months' history of severe cough and slight pains. All the cysts were removed radically and all the patients were alive and well, three months to seven years after the operation.

UNDEFINED CYSTS

Our series contains 4 cases with cystic affections in the upper anterior mediastinum, where we have not been able to classify the specimens definitely,

in spite of the fact that the histologic material was rather extensive. One of these specimens, a grapefruit-sized multicystic formation removed from a man aged 58, could possibly be a so-called "burned out thymoma" (Blalock[8]). This patient had no signs of myasthenia gravis at admission or in the history. In 2 other cases, the cysts, about the size of a hen's egg, had a thick fibrous wall with cholesterin granulomas but without any epidermal or other organoid structures. However, we cannot exclude the possibility that they are adult teratomas of the "dermoid cyst" type with strong regressive changes. The last specimen, large as a mandarin orange, had a thin, fibrous wall with rather abundant lymphocyte infiltrates but lacked entirely epithelial or endothelial lining.

SUMMARY

This is a survey of 155 patho-anatomically verified primary mediastinal tumors and cysts, treated at the Clinic of Thoracic Surgery at Sabbatsberg Hospital during the period 1944 to 1954. Table I gives the composition of the material, and on page 458 the mediastinal affections, excluded from or missing in the series, are recorded. A comparison between the composition of this series and those of some previous investigators is given in Table II.

Not less than 61 per cent of these tumors and cysts were discovered by mass radiography, and Fig. 1 gives the relation between the frequency of mass radiography in Sweden and the number of growths in this series, thus discovered.

Location, gross anathomy, histology, age and sex distribution, clinical course, and the result of follow-up are recorded.

This series includes a number of rare tumors, some being difficult to classify and the investigation has in certain details given results, partly new and partly disagreeing with previous experience. Thus, all the 3 cases of ganglioneuroblastoma ran a benign course, which does not tally with the findings of, e.g., Ackerman and Taylor.[1] Among the hemangiomas (recorded on page 468) there is 1 case of hemangiopericytoma and another peculiar melanin-pigmented specimen, possibly a neurovascular malformation. The series includes 1 case of plasmacytosarcoma with no signs of multiple myeloma.

All tumors classed as thymomas have been diagnosed in accordance with the restrictive definition given, among others, by Seybold and associates,[48] and in no case were there signs of extrathoracic metastasis. Only 32 per cent of the thymoma patients had myasthenia gravis (Seybold and co-workers[48]; 75 per cent).

The material includes 2 tumors, quite unconnected with the lung or bronchial tree but with a structure very similar to that of bronchial adenoma (Figs. 12 and 13). In another similarly located, infiltratively growing tumor the microscopic picture was a malignant counterpart of the former (Fig. 14). The possibility is emphasized that these tumors are teratomas with a one-sided development, arising from the bronchial anlage. We have not been able to find any identical case in the literature.

Besides 19 cases of classical bronchial cyst, the series includes 2 multicystic specimens located in the costovertebral angle and with a structure similar to a

lung anlage (Figs. 16 and 17). The occurrence of such mediastinal malformations is in consonance with the theory that bronchial cysts develop from a pulmonary anlage. We have not seen any identical cases in the literature.

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FIBROUS DYSPLASIA OF BONE: ANALYSIS OF 15 CASES OF SURGICALLY VERIFIED COSTAL FIBROUS DYSPLASIA

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FIBROUS dysplasia of bone is characterized by the occurrence of one discrete skeletal defect, or of a few, or numerous such defects. Brown-to-yellow patches of cutaneous pigmentation may accompany the bone lesions, especially in severe disseminated fibrous dysplasia. When, in addition to cutaneous pigmentation, patients having polyostotic fibrous dysplasia show signs of endocrine abnormalities, especially precocious puberty among girls, the condition is commonly referred to as "Albright's syndrome."[1] Excluding the 67 cases collected in the armed forces,[2] no sex predilection of fibrous dysplasia has been observed. The etiology of these bone defects is unknown; the weight of evidence indicates that they are due to a congenital anomaly in which the activity of the specific bone-forming mesenchyme is perverted.[3] The cause of the extraskeletal lesions is equally obscure. The disease usually appears early in life and progresses most actively during the patient's growth. The long-standing confusion of polyostotic fibrous dysplasia with the bone lesions of hyperparathyroidism should rarely be a problem when one considers the diffuse demineralization, chemical changes in blood, renal lithiasis, and the older average age of patients having hyperparathyroidism.

When a single bone only is involved, the lesion is called "monostotic," even though the defects occasionally are multiple. It is difficult to reach an accurate figure concerning the relative frequency of the monostotic as compared to polyostotic lesions. The literature is likely weighted in favor of the more dramatic full-blown examples of Albright's syndrome. Furthermore, total skeletal roentgenograms would be necessary to establish the authenticity of the lesions reported as monostotic. The consensus is that the solitary lesions are more common. Berger and Jaffe[4] have said that for each patient having fibrous dysplasia with full-blown Albright's syndrome there are 20 or 30 patients who have fibrous dysplasia with only one or a few bones involved.

The cutaneous pigmentation is owing to an increased deposition of melanin in the basal layer of the epithelium. These single or multiple brown-to-yellow discrete areas may be of any size. In the review of 256 cases by

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Pritchard,[5] pigmentation was recorded in 79 instances, although it was not mentioned in any of the 67 monostotic cases contributed by Schlumberger.[2] Pigmentation is much more likely to occur with severe polyostotic fibrous dysplasia, especially if endocrine abnormalities are present, than with the monostotic form, although it has been observed in association with the latter form.

Endocrine effects occur much more commonly among women than among men with polyostotic fibrous dysplasia. Precocious sexual development was observed in 37 women and 5 men, or 16.4 per cent of the series reviewed by Pritchard. Premature menstruation, early development of secondary sexual characteristics, and premature fusion of epiphyses resulting in adults of small stature are the common features. Rarely are retardation of puberty and hypogenitalism[6, 7] exhibited in either sex. Hyperthyrodism seems to occur with extraordinary frequency among patients with polyostotic fibrous dysplasia, a total of 6 unequivocal examples of this association having been observed by 1951.[8]

A rather special type of fibrous dysplasia occurs in jawbones[4] and in bones at the base of the skull. The fibrous tissue of lesions in these regions, especially in the skull, tends to show much more metaplasia to bone than it does in other sites. This often produces roentgenologically dense, expanding lesions that are more or less heavily calcified. Many lesions of this type have been called "ossifying fibromas" and even "osteomas." The very appropriate term, "fibro-osseous dysplasia," has been suggested in these cases. Berger and Jaffe consider that in about 10 per cent of the cases of monostatic fibrous dysplasia, the lesions occur in bones of the head, and one-half of the patients with polyostotic fibrous dysplasia will have one or more lesions in bones of the skull. Much evidence indicates, furthermore, that the condition included under the descriptive term "leontiasis ossea"[9] is due to fibrous dysplasia of bones of the skull and face. Some workers[10] also consider "cherubism" to be a manifestation of fibrous dysplasia.

Any bone may be affected in fibrous dysplasia. Several authors have stressed the occasional tendency for the polyostotic lesions to be confined to one side of the skeleton. In the collected series of all types of fibrous dysplasia reviewed by Pritchard, he tabulated the distribution of lesions as follows: lower limbs, 118 cases; upper limbs, 87; skull, 52; face, 31, and trunk bones, 49. Schlumberger noted that a rib was the site of involvement in 29 of 67 isolated lesions diagnosed as fibrous dysplasia of bone. More recently Hochberg[11] has found that, including Schlumberger's group of 29 cases of fibrous dysplasia, the total of 35 cases comprised one-third of all cases of benign costal tumors which he found reported in the literature.

The relatively high frequency of costal involvement in both monostotic and polyostotic fibrous dysplasia makes this lesion of significance to the general surgeon. The lesion is often asymptomatic and discovered on routine roentgenograms of the thorax, whereupon a decision regarding therapy must be made. For these reasons a study of our surgically verified costal lesions of this type was undertaken.

PRESENT SERIES

A review of the series of surgically treated costal tumors at the Mayo Clinic showed 15 examples of fibrous dysplasia. Thirteen of these lesions were excised intact along with the involved segment of rib, and curetted fragments were obtained in the other 2. The markedly selected nature of these cases limits their value in understanding fibrous dysplasia as a whole. In most cases of polyostotic fibrous dysplasia, costal lesions have not been treated surgically. For example, in the report on Albright's syndrome from the Clinic by Dockerty and associates,[12] 4 of 6 patients whose cases were presented had costal lesions which were not subjected to operation. The surgically excised costal lesions afford a unique opportunity, however, to study intact gross specimens, since the average fibrous dysplasia of other bones is usually excised piecemeal with a curette if removed at all.

Age and Sex.—The ages of the 15 patients varied from 18 to 65 years, and 12 of them were more than 30 years of age. The relatively advanced age of this group is partially explained by the fact that the lesions were accidental roentgenographic discoveries in 7 instances. Several of the other patients had had symptoms for a long period. Eight patients were women and 7 were men.

Clinical Features.—Three patients had noted some thoracic discomfort for periods of ten to fifty years; 5 had had thoracic pain or soreness for periods ranging from one week to four years, and 7 had not had any symptoms. Pathologic fracture had occurred in only 1 case.

Physical examination was noncontributory in 10 cases, although in 1, the patient had had multiple subcutaneous lipomas over the surface of the thorax and abdomen. Three patients had palpable enlargements or irregularities of the affected ribs and local tenderness was present in 2. Patches of cutaneous pigmentation were not recorded in any case of this series.

Roentgenographic Findings.—A frequent manifestation of fibrous dysplasia of the ribs consists of moderate expansion of a long segment of the rib with the affected bone having a uniform ground-glass appearance so that the cortex and spongiosa cannot be distinguished. This type of fibrous dysplasia does not have a cystic appearance and often more than one rib is affected. The correct diagnosis is usually quite obvious and the patients seldom undergo operation for the condition. For that reason no patients having lesions of this type are included in the group that underwent operation.

The roentgenograms revealed the bony defect in each case. The characteristic lesion produced expansion of the rib with an irregularly attenuated, though intact, cortex. The expansion was usually toward the thoracic cavity so that even when the lesions were large the tumor often was not evident externally. The expanded portion was basically radiolucent but frequently appeared trabeculated and showed a fuzzy, patchy type of density. The ends of the affected zones were sharply demarcated from the uninvolved portions of the rib. The ribs with long zones of fibrous dysplasia became distorted in contour and expanded to variable degrees so that the normal parallelism to adjacent ribs was lost. In lesions with classic roentgenologic findings, the diagnosis of fibrous dysplasia can be made with considerable certainty. In

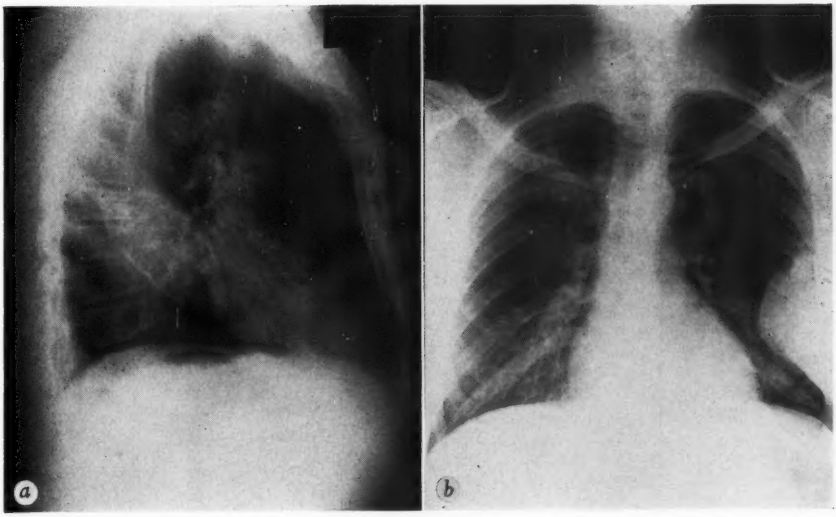

Fig. 1, *a* and *b*.—Fibrous dysplasia of the seventh left rib as seen in the posteroanterior and lateral roentgenograms.

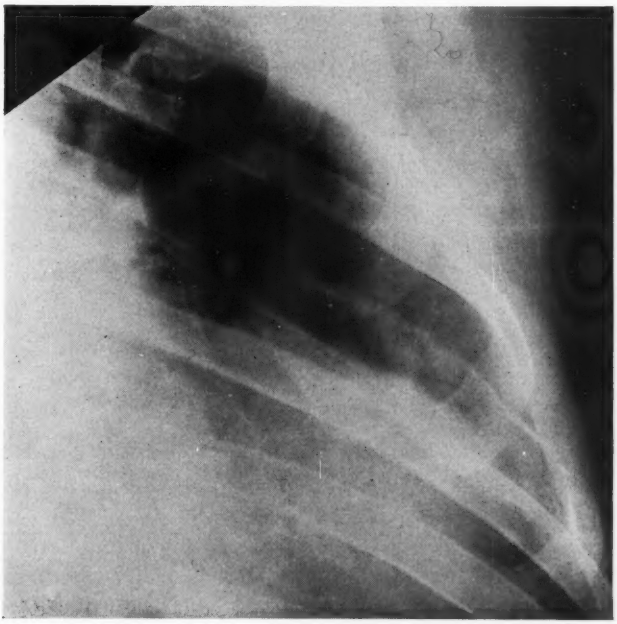

Fig. 2.—Fibrous dysplasia of long segment of left fifth rib.

some cases, however, the roentgenologic defects may be mistaken for lesions such as central cartilaginous tumors, or myeloma, or eosinophilic granuloma (Figs. 1 and 2).

Fig. 3.—Surfaces produced by longitudinal section of the lesion shown in Fig. 1, *a* and *b*.

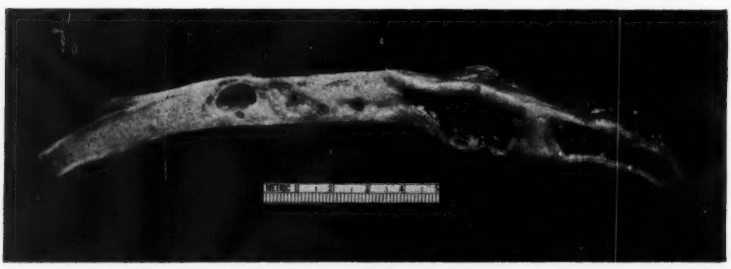

Fig. 4.—Only slightly expansile, partially cystic fibrous dysplasia of left fifth rib (same case as Fig. 2).

Fibrous dysplasia was apparently polyostotic in 3 of the 15 patients. One patient whose diagnosis was verified by biopsy had similar defects in two other ribs and a radiolucent area in the ilium. Another patient with expanding lesions of a rib and the transverse process of a lumbar vertebra was thought to have myeloma until the affected rib was curetted for biopsy. One patient with similar defects in two ribs had 1 of the lesions removed.

Pathologic Aspects.—The specimens removed (Figs. 3 and 4) varied in size from 5 by 1 by 1 cm. to 19 by 6 by 5 cm. They were uniformly central, expanding, fibrous masses. The expanded costal cortex showed irregular thinning, and its internal surface frequently exhibited corrugations, the shadows of which sometimes appeared as septa in the roentgenograms. Partially calcified osteoid masses in the fibrous tissue of 10 lesions imparted a gritty character to the cut surface. Cystic areas, which measured as much as several centi-

Fig. 5.

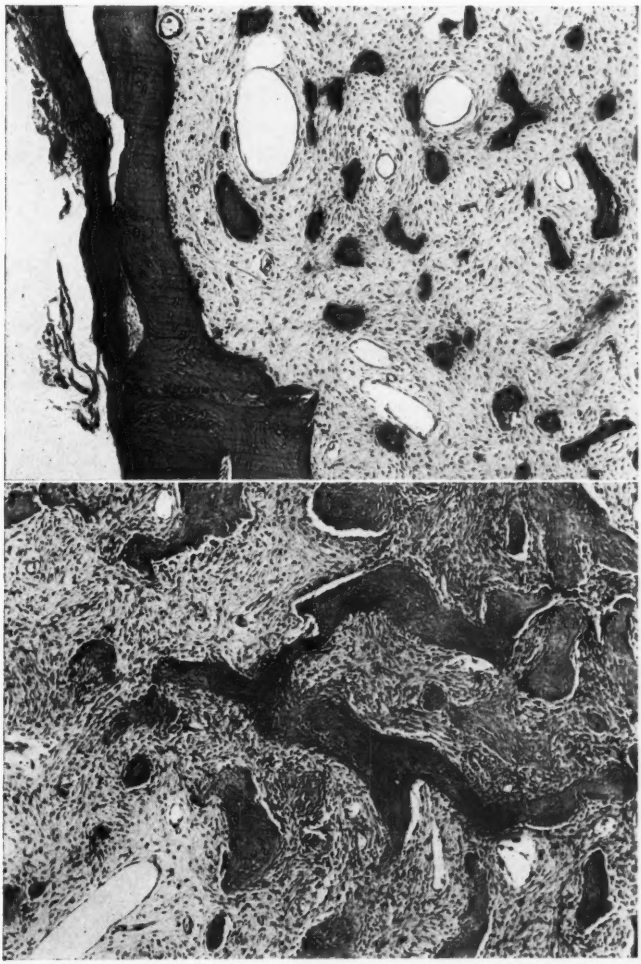

Fig. 6.

Fig. 5.—Typical fibroblastic tissue containing irregular islands of new bone. The broad vertical band near one end represents the distended cortical shell. (Hematoxylin and eosin; ×75.)

Fig. 6.—More cellular area of fibrous dysplasia with larger osteoid masses. (Hematoxylin and eosin; ×75.)

meters in diameter in the larger lesions, were present in 6 cases. In 2 instances, the cysts comprised more than one-half the bulk of the area of dysplasia. Three specimens contained brown zones owing to deposits of hemosiderin. Although fusiform expansion was the usual gross configuration, 1 lesion involved 17 cm. of a rib but the rib had expanded to a maximum of only 1.5 cm.; another rib was distinctly dumbbell-shaped with two globular expansions connected by an isthmus of fibrous tissue. In all cases, the dysplastic tissue was distinctly demarcated from the adjacent normal bone.

The histologic features of fibrous dysplasia have been well documented. Briefly, the findings in this series (Figs. 5 and 6) were as follows: fibroblasts lying in a more or less dense collagenous matrix dominated the picture. The cellularity varied greatly from lesion to lesion and to a less extent in different portions of the same lesion. All lesions contained zones in which irregular osteoid masses had been laid down in the fibrous matrix. Slight to moderate calcification of these osteoid trabeculae usually had occurred. The amount of osteoid trabeculae varied considerably, and in several lesions central areas contained little or none. As pointed out by Schlumberger, these trabeculae which aid greatly in making the diagnosis tend to be most prominent in the peripheral zones. Cartilaginous zones were not found in any of the lesions. Focal collections of hemosiderin, usually within macrophages, were found in 7 lesions and, as already noted, they were prominent enough to produce grossly visible brown zones in 3. Benign multinucleated cells were present in 8 specimens and in several instances they were numerous enough in foci to simulate giant-cell tumor of bone superficially. Myxomatous changes were found in parts of the fibrous tissue of 7 of these costal tumors. This degenerative change apparently preceded cystic formation because it was especially prominent in the vicinity of gross and microscopic cysts. Inflammatory cells were rare; foci of lymphocytes and plasma cells were found in 1 lesion.

COMMENT

This series of cases of fibrous dysplasia of ribs emphasizes some of the features of this peculiar growth anomaly. Cystic formation may be a prominent pathologic characteristic of the gross specimen, and in rare instances, the dysplastic zone may be predominantly cystic. Although chondroid foci within the lesional tissue have been considered to give an important diagnostic clue in many cases of fibrous dysplasia,[13] their complete absence in this series is noteworthy.

The indolent nature of the pathologic process in this disease is suggested by the fact that no symptoms had been produced in 7 patients and several of the remaining 8 had had but slight difficulty for many years.

Once one is familiar with this entity the diagnosis is rarely difficult. The metaplastic osteoid trabeculae have a characteristic appearance and are not found in nonosteogenic fibroma. The lack of cellular anaplasia precludes the serious consideration of any malignant process. The collagen production in fibrous dysplasia contrasts with the lack of such production in diagnostic fields

of a giant-cell tumor of bone, and this fact alone should differentiate the two conditions. The lesions with large cystic areas may cause some confusion, but even in these the central expansile character of the process as observed roentgenologically and grossly, coupled with histologic evidence from the solid areas, ordinarily solves the problems.

Regarding treatment of fibrous dysplasia of ribs, there may be some disagreement. In examples of the polyostotic form of the disease, costal lesions rarely will require treatment. With single or only a few lesions, the physician may not be absolutely certain of the diagnosis without pathologic study. In such cases, a plea should be made for total excision of the involved regions if at all feasible. Such surgical management would help to prevent contamination of the wound by malignant tissue if the lesion proves to be malignant. The size of the lesions of several of our patients constituted adequate indication for removal. Malignant change in fibrous dysplasia has been observed so rarely[14, 15] that this threat can scarcely be considered an indication for surgical intervention.

SUMMARY

Fibrous dysplasia of bone is characterized by the occurrence of one, a few, or numerous discrete skeletal defects. The lesions most commonly are limited to one bone in which case the condition is known as the "monostotic" type. However, there are cases in which disseminated skeletal lesions have been associated with endocrine abnormalities and areas of pigmented skin. The etiology of the bone defects is unknown; however, the weight of evidence indicates that the defects are caused by a congenital anomaly in which the activity of bone-forming mesenchyme is perverted. The extraskeletal lesions have an obscure etiology also. It is a disease of early life and progresses most actively during the patient's growth. Fibrous dysplasia may affect any bone. The frequency of costal involvement is relatively high, which makes this lesion significant to the general surgeon. The lesion is often asymptomatic, discovered on a routine roentgenogram of the thorax, and a decision regarding therapy must be made. For these reasons a study of our surgically verified costal lesions was undertaken.

Our study included 15 cases of fibrous dysplasia of ribs. The ages of the patients ranged from 18 to 65 years. Eight patients were women and 7 were men. Clinical signs and symptoms were few. Three patients complained of thoracic discomfort, 5 of thoracic pain or soreness of a mild nature, and 7 were free of symptoms. Ten patients had no physical signs, 2 had local soreness, and 3 had noted some enlargement over the affected rib. A roentgenogram of the thorax revealed the lesion in each case. The lesions were found to be uniformly central, expanding fibrous masses, ranging in size from 5 by 1 by 1 cm. to 19 by 6 by 5 cm. The expanded cortex of the rib showed irregular thinning. The internal surface of the cortex often exhibited corrugations, the shadows of which appeared as septa in the roentgenograms. Partial calcification makes the lesion gritty and cystic areas were found in 6 cases. In all cases the dysplastic tissue was distinctly demarcated from the adjacent normal

bone. The main histologic feature is fibroblasts lying in a more or less dense collagenous matrix. The pattern is irregular, however, since the cellularity varies and regions of osteoid trabeculae and calcification may be found.

The diagnosis of this condition is not difficult if one is familiar with it but some disagreement exists regarding therapy. In examples of the polyostotic form, costal lesions rarely require treatment. If only a single lesion or a few lesions are present, pathologic study may be necessary before a positive diagnosis can be made. Therefore, a plea is made for total excision of the involved portion if at all possible.

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HEREDITARY HEMORRHAGIC TELANGIECTASIA

Report of a Case With Hemothorax

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MASSIVE hemothorax in a patient known to have hereditary hemorrhagic telangiectasia (Osler-Rendu-Weber disease) has been previously reported only once. We encountered this complication in a 44-year-old man. Its rarity and the difficult problems involved in its management have prompted this report.

The first description of hereditary telangiectasia was written in 1865 when Babington[1] gave an account of recurrent epistaxis through 5 generations of a family. The cases reported by Sir William Osler[1-3] established the disease as a definite entity. During the past fifty years, hundreds of case reports have appeared and more than 150 families have been described. Bleeding from practically every organ has been reported in this disorder. Other diseases have been described in association with hereditary telangiectasia but pulmonary arteriovenous fistula is the only associated disorder commonly seen. More than one-half of the pulmonary arteriovenous fistulas reported to date have been in association with hereditary telangiectasia.

The name, hereditary hemorrhagic telangiectasia, first used by Hanes[4] in 1909, describes three of the criteria for diagnosis. The triad of telangiectases of the skin, mucous membranes, or viscera; a tendency to bleed from these lesions; and a family history of intermittent hemorrhages is strong evidence for the disease. If, in addition, the patient has a normal clotting mechanism, the diagnosis is established. The chief obstacle to diagnosis is the failure of the physician to think of the disease. These patients almost invariably complain of epistaxis which usually begins near puberty and recurs frequently thereafter. Blood dyscrasias are easily ruled out by laboratory studies. A careful search of skin and mucous membranes will reveal the lesions. Bronchoscopy,[5] gastroscopy, or proctoscopy[6] may show telangiectases of the trachea, bronchi, stomach, or rectum.

The lesions of hereditary telangiectasia may be located on the surface or in the parenchyma of any organ. The visible telangiectases may be pin-point and flat but are usually several millimeters in diameter and slightly elevated. Their color varies from cherry red to purple. Those on the mucous membranes and skin usually appear near the age of puberty but have actually been noted

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at birth. Spider-like telangiectases are also frequently present. The absence of pulsation distinguishes these spider angiomas from those seen in liver disease.[7] Bleeding from viscera usually does not occur before the third decade of life. There is a tendency for new telangiectases to appear with increasing age. These nodular lesions blanch readily and may pulsate. Microscopic study shows vascular sinuses of irregular size and shape, lined by a thin layer of endothelium. Muscular and elastic coats are absent. Because of the thin walls and proximity to the surface, these sinuses bleed readily with slight trauma and, because of the lack of contractile elements in the wall, bleeding may not cease spontaneously.

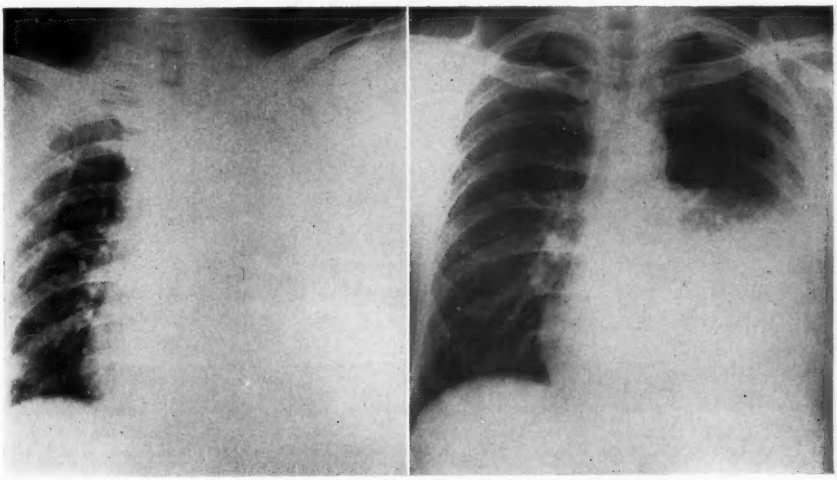
Fig. 1.—Typical lesions of hereditary hemorrhagic telangiectasia on lips and tongue.

Descriptions of the pulmonary arteriovenous fistulas found in this disease show them to be anatomically similar to the peripheral telangiectases. They are large sinuses interposed between arteries and veins. The afferent artery and efferent vein are usually dilated and thin walled. The danger of massive hemorrhage into the pulmonary parenchyma is ever present. In over one-half of the cases, the pulmonary arteriovenous fistulas are multiple. Goldman[8] believes that all congenital pulmonary arteriovenous fistulas are a localized manifestation of generalized hereditary telangiectasia.

Hereditary telangiectasia is transmitted as a simple dominant trait and affects both sexes. The children of a family in which one parent is affected, therefore, have a 50 per cent chance of inheriting the disease. Often the lesions are confined to the mucous membranes of the nose and mouth and epistaxis may be the only symptom (Fig. 1). In general, the earlier in life the telangiectases make their appearance the worse the prognosis.

The only known report of a child born to parents who were both afflicted with hereditary telangiectasia described the appearance of new telangiectases daily until death at less than 3 months of age.[9] The probability of such a lethal form of the disease in a child of parents who are both affected is one in four.

The predominant symptom of hereditary telangiectasia is recurrent painless hemorrhage. Epistaxis is such a consistent manifestation that one should question the diagnosis if the patient has no history of epistaxis. When bleeding is from a visible nodular lesion it may be seen to come in spurts. Bleeding may occur in the mouth, nose, stomach, bowel, kidney, bladder, liver, spleen, brain, or meninges. Some have noted an increase in size of the telangiectases and tendency to hemorrhage during menstrual periods.[10] Campbell[11] suggests that many cases of "essential hematuria" are really due to hemorrhagic telangiectasia. We suspect that the same is true of "spontaneous hemothorax." Massive painless gastrointestinal bleeding is very common.[12] The bleeding may lead to signs of anemia but this is frequently masked by the polycythemia which is an accompaniment of arteriovenous fistulas. Patients may, therefore, have a normal or high hemoglobin even after a hemorrhage.[13] Others alternate between polycythemia and a marked anemia.[14] Cyanosis, shortness of breath, clubbing of fingers, and a bruit are common in this group. Fatal hemoptysis from such a fistula has been reported.[15]



A. *B.*

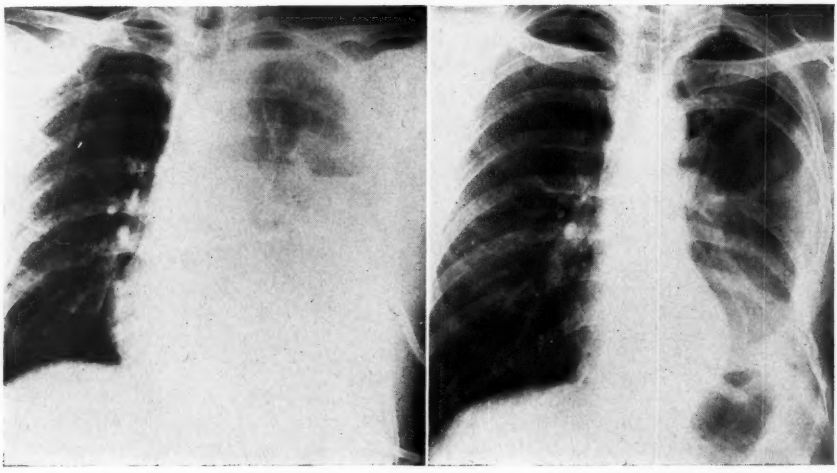
Fig. 2.—*A*, Roentgenogram on admission to another hospital showing opacity of left hemithorax of fluid density with slight displacement of the heart to the right. *B*, Roentgenogram on admission to Veterans Administration Hospital, McKinney, Texas, showing opacity of lower half of left chest with curved line typical of fluid.

Treatment with rutin, irradiation, estrogens, ascorbic acid, moccasin venom, vitamin K, and other drugs has given poor results. Oral thrombin appeared to be effective in a case of massive bleeding from multiple telangiectases of the small bowel.[16] Because of the tendency for remissions and exacerbations in the hemorrhagic tendencies the evaluation of any therapy is difficult.

CASE REPORT

The patient was a 44-year-old laborer, who had a sudden onset of shortness of breath and left chest pain on Feb. 5, 1952. He was admitted to a hospital two days later with a

hemoglobin of 9.8 Gm. and a thoracentesis yielded 250 c.c. of blood. Thoracentesis was performed the following day and 600 c.c. of blood removed. He was transferred to the Veterans Administration Hospital, McKinney, Texas on Feb. 13, 1952, with a diagnosis of "hemothorax, left, probably due to neoplasm of the pleura." Upon arrival the patient was acutely ill, pale, listless, with temperature 101.5° F., pulse 102, and blood pressure 105/70. There were signs of fluid in the left chest and the hemoglobin had dropped to 7 Gm. He had multiple small elevated cherry-red telangiectases on the tongue, buccal mucosa, lips, nasal mucosa, and skin. The skin telangiectases were most numerous over the face and trunk but there were some over his entire body as far distally as the ankles. He gave a history of troublesome nosebleeds several times weekly for as long as he could remember, one episode of tarry stools of two weeks' duration at age 14, and frequent bleeding from the gums. A sister had similar lesions but had no serious hemorrhage.



A. *B.*

Fig. 3.—*A*, Roentgenogram after repeated thoracenteses, intercostal catheter drainage, and use of Varidase showing opacity of left chest with multiple air-fluid levels due to loculated hemothorax. *B*, Roentgenogram over 7 months after decortication showing clearing of left hemithorax but the left diaphragm remains elevated and the costophrenic agle obliterated.

At thoracentesis on Feb. 15, 1952, 1,600 c.c. of blood was removed. He was given 1,000 c.c. of blood immediately, and 500 c.c. the following day. Bronchoscopy was negative. On the fourth day, a thoracentesis yielded 1,100 c.c. of blood and resulted in roentgen evidence of some clearing of the effusion but a day later there was a reaccumulation of the fluid (Fig. 2). On February 22, an intercostal catheter was inserted through the ninth intercostal space in the left posterior axillary line and 3,200 c.c. of blood removed. Thereafter, it drained about 500 c.c. of blood daily and he was supported by multiple transfusions. The intercostal catheter became plugged frequently and small amounts of Varidase were used on several occasions to clear it. This therapy was abandoned because it seemed to perpetuate the bleeding. X-rays, on February 29, showed a loculated hemothorax (Fig. 3).

On March 5, a left thoracotomy was performed and no evidence of tumor found. Decortication of the clotted hemothorax was carried out and it was noted that there was excessive bleeding from many tiny areas of the pleura. A total of 4,000 c.c. of blood was required during and immediately following surgery. At this time the bleeding time, clotting time, platelet count, clot retraction, prothrombin, Factor 5, and prothrombin utiliza-

tion were normal. Coombs tests direct and indirect were negative. Fibrinolysin was negative after five and one-half hours and a test for circulating anticoagulant was also negative.

The patient drained large amounts of bloody fluid via the intercostal catheter but the lung failed to re-expand completely. On March 19, he began having a septic fever and a culture of the pleural fluid showed hemolytic staphylococcus. On April 2, a rib resection was done and a large empyema pocket evacuated. His course was one of steady improvement from that time on, and on May 20, he received his twenty-fifth and final transfusion. He had frequent expistaxis while in the hospital, but his chest symptoms gradually improved. The lung slowly re-expanded although he still had slight drainage at the time of his discharge. He has been followed for three years and has had epistaxis and hematuria but no recurrence of hemothorax.

DISCUSSION

In the case of spontaneous hemothorax reported by Armentrout and Underwood,[17] the diagnosis of hereditary telangiectasia is undeniable. The patient at the age of 25 apparently had hemothorax because "her physician found signs of fluid in her chest and aspirated blood from her left thorax in an amount estimated by her to be about one and one-half quarts. Convalescence from this illness was uneventful and she had no recurrence." Our case is the second one of hemothorax reported in a patient known to have hereditary telangiectasia. His prolonged illness is in marked contrast to the uneventful recovery of the patient just mentioned. We now believe that the many small but vigorously bleeding areas encountered in the pleura at the time of decortication represented small telangiectases. Extensive scarring prevented their definite identification as such at this time. Prompt use of intercostal catheter water-seal drainage with a slight negative pressure would be more likely to control the bleeding and prevent a clotted hemothorax than would repeated aspirations or surgical intervention. We would be reluctant again to attempt decortication in such a patient during the active bleeding phase. However, decortication was the logical treatment in this case because localized arteriovenous fistula or tumor could not be ruled out and there was present a partially clotted, loculated, infected hemothorax.

There have been two reports of hemothorax from arteriovenous fistulas in patients who apparently showed no extrapulmonary lesions of hereditary telangiectasia.[18] The telangiectases may have been unrecognized or considered unimportant. One of these patients died at the age of 23 from hemothorax and was shown to have multiple pulmonary arteriovenous fistulas. The other, reported by Bowers,[19] died of hemothorax on the second day of life and was found to have multiple pulmonary arteriovenous fistulas.

Whether all pulmonary arteriovenous fistulas are manifestations of hereditary telangiectasia is open to question. In many of the original descriptions of pulmonary arteriovenous fistulas, the peripheral lesions of hereditary telangiectasia are not mentioned. They may have been unrecognized or considered irrelevant. Only in recent years has the relationship between the two lesions been recognized. Cardiologists and thoracic surgeons are now aware of the frequent coexistence of pulmonary arteriovenous fistulas and hemor-

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rhagic telangiectasia. In most recent reports of pulmonary arteriovenous fistulas, note has been made that the patients were known to have hereditary telangiectasia.[13, 15, 17, 18, 20-26]

SUMMARY

This is the second reported case of spontaneous hemothorax in a patient with hereditary hemorrhagic telangiectasia. Treatment consisted of thoracentesis, intercostal catheter drainage with Varidase instillation, and later decortication. The likelihood of recurrent pleural hemorrhage from multiple small arteriovenous fistulas makes the treatment of hemothorax particularly difficult in this disease. Surgical intervention should probably be reserved as a last resort, or in the treatment of complications of a clotted hemothorax.

The occurrence of pulmonary arteriovenous fistulas in patients with hereditary telangiectasia is so common that chest x-rays should be a part of the routine work-up of these patients.

We suspect that hereditary hemorrhagic telangiectasia could be diagnosed in many cases of "spontaneous hemothorax" by a careful history and search for the lesions.

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MASSIVE CYSTIC PULMONARY HAMARTOMA

Report of Two Cases

Robert C. Jackson, M.D.,* John R. McDonald, M.D.,** and
O. Theron Clagett, M.D.***
Rochester, Minn.

PULMONARY hamartoma has been found in 0.25 per cent of a series of consecutive necropsies.[1] Most of these tumors are small and are unrecognized in the ordinary roentgenogram of the thorax. Many of the smaller ones are interpreted as Ghon tubercles. When a hamartoma attains a size greater than 1 cm., it becomes a diagnostic problem. A patient with such a problem usually undergoes diagnostic thoracotomy. A hamartoma removed under these circumstances is seldom larger than 5 cm. in diameter. In two of the hamartomas of the lung recorded in the files of the Mayo Clinic, the most unusual feature was the large size. These two cases form the basis of this report.

BACKGROUND INFORMATION

In 1904, Albrecht[2] originated the term "hamartoma," derived from the Greek words meaning "error" and "tumor." Translated, his definition was "tumorlike malformations in which occur only an abnormal mixture of the normal constituents of the organ in which they are found. These malformations may consist of a change in the quantity, arrangement, degree of differentiation, or any combination of these. The conclusion to be drawn from histologic examination is that they originated in abnormal mixing of tissues during their development." Albrecht was referring to specific tumors of the spleen, liver, and kidney.

In 1922, Feller[3] initiated use of the term "hamartoma" in conjunction with certain benign cartilaginous pulmonary lesions, but only recently has the application of this term to these lesions been generally accepted. Until the past decade, most cases were recorded as chondroma of the lung, despite frequent recognition of their complex tissue structure since the nineteenth century.[4]

Pulmonary hamartoma occurs more frequently in males, in a ratio of 2 or 3 to 1. The range in age is from prematurity to old age, but those reported in childhood are few in number, probably because of the dearth of necropsy material and the relative infrequency of routine roentgenographic examina-

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tion of the thorax in this age group. In a series of 156 indeterminate solitary circumscribed pulmonary lesions reported by Hood and co-workers,[5] 16 per cent proved to be hamartomas. No significant predilection for a given lobe or lung has been noted.

Clinically, hamartomas are frequently asymptomatic and are discovered by routine x-ray examination. Cough or thoracic pain may occasionally be the presenting symptom. Endobronchial hamartomas commonly produce wheezing and symptoms of pulmonary suppuration. Ten per cent are said to arise endobronchially, but this figure is derived from collected series and is undoubtedly influenced by the tendency to report lesions in unusual sites. In our experience, true chondromas occur more commonly in this position than do pulmonary hamartomas.

A roentgen shadow of a sharply circumscribed, peripheral pulmonary lesion with lobulated margins and clear adjacent pulmonary tissue should suggest the possibility of hamartoma. The demonstration of diffuse punctate areas of calcification in such a lesion makes the diagnosis probable. Lemon and Good[6] have emphasized the inadequacy of roentgenograms in establishing a definitive diagnosis, and we firmly believe that surgical exploration is indicated in all patients if their general condition permits.

At thoracotomy, many hamartomas are readily recognizable in situ. They are characteristically less than 4 cm. in diameter, spherical or ovoid, subpleural, sharply demarcated from pulmonary parenchyma, of firm cartilaginous consistency, and have a white nodular surface. Diagnosis should be made by frozen-section technique, thereby enabling maximal conservation of functioning lung. The majority are readily enucleated or removed by wedge resection. Occasionally they lie deeply imbedded in hilar structures, necessitating more extensive resection. Endobronchial lesions usually require major resection, although the surgeon should be alerted to the possibility of bronchotomy with conservative excision of the tumor and bronchial reconstruction, provided the associated suppurative changes are of a reversible nature. Tissues of both mesodermal and endodermal origin are present in pulmonary hamartomas. There is considerable variation in microscopic appearance. Usually, hyaline cartilage is present in abundance, with intervening clefts lined by columnar or cuboidal epithelium which may be ciliated or mucus-producing in part. Fibrous and myxomatous connective tissue and fat are often present, and more rarely elastic or fibrocartilage or smooth muscle may be identified. Calcified areas are frequently present in the cartilage, and occasionally bone formation occurs. None of the afore-mentioned tissues need necessarily be present to make a diagnosis of hamartoma. In some of these lesions, areas may be found which are remarkably similar to normal bronchi. Derangement of architecture, overabundance of fat, and occasionally a suggestion of embryonal tissue will be noted, however. This feature is illustrated in Case 2.

CASE REPORTS

CASE 1.—A 54-year-old woman was referred for treatment of a pulmonary cyst. She had complained of moderate exertional dyspnea for four years, and of mild orthopnea that

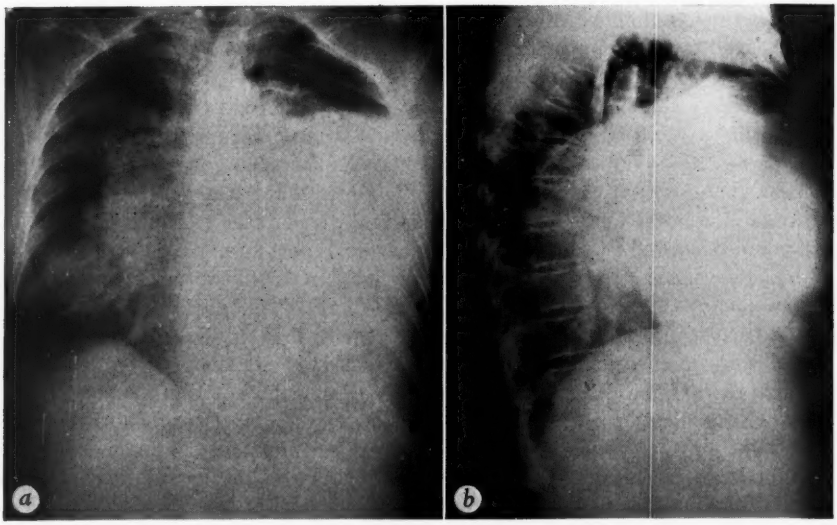

Fig. 1, *a* and *b*.—Case 1. Posteroanterior and left lateral roentgenographic views of the thorax showing hamartoma.

Fig. 2.—Case 1. Gross specimen of hamartoma with liquid contents evacuated.

was relieved by lying on her left side. Five times in the past three and one-half years her local physician had given her temporary relief by thoracentesis, which was productive of 1 to 1½ quarts of reddish-brown fluid.

Significant physical findings were the presence of all heart sounds to the right of the sternum and no evidence of functioning lung in the lower two-thirds of the left side of the thorax. The superficial veins of the neck were distended, particularly during inspiration. Laboratory data were within the normal range. Posteroanterior and lateral roentgenographic views of the thorax demonstrated a well-defined opacity in the lower three-fourths of the left hemithorax (Fig. 1). The heart and other mediastinal structures were markedly displaced to the right.

Left thoracotomy revealed a large, lobulated, cystic tumor occupying fully two-thirds of the left hemithorax. It involved most of the left lower lobe and was densely adherent to the pericardium, thoracic wall, lung, and diaphragm. During the extensive dissection necessary for removal of the lesion, its wall was ruptured, liberating 2,800 c.c. of watery brown fluid. After repair of many small bronchial fistulas, the lung expanded well. Two thousand cubic centimeters of whole blood was given during the procedure. On the third postoperative day, coma developed and the neurological findings suggested a right cerebrovascular accident. Death occurred on the fourth postoperative day. Permission for necropsy was not granted.

Fig. 3.—Case 1. Representative section from hamartoma. (Hematoxylin and eosin; ×50.)

The surgical specimen measured 17 by 15 by 12 cm. and weighed 600 grams without its fluid contents (Fig. 2). The surface was nodular and grayish white. A large single cyst occupied the center of the mass. The wall varied from 0.4 to 5.5 cm. in thickness and its inner surface was partly smooth, but there were many roughened areas where the tissue appeared to be necrotic. On cut surface the tumor appeared grossly to consist of hyaline cartilage with small scattered areas of dense calcification.

Microscopically, there was abundant hyaline cartilage with some areas of calcification (Fig. 3). Much adult fat and fibrous connective tissue were present and there were small cystic spaces lined with flattened and cuboidal epithelium. Part of the tissue was necrotic, and the presence of hemosiderin suggested previous hemorrhage. The large cyst was lined with cuboidal epithelium in the smooth areas.

CASE 2.—A 27-year-old man was admitted for evaluation of a pulmonary cyst known to have been present for eleven years. He denied having any pulmonary symptoms. No unusual findings, except decrease in the breath sounds over the right part of the thorax, were revealed by a thorough physical examination.

Laboratory data were normal. Posteroanterior and lateral roentgenographic views of the thorax demonstrated a well-circumscribed cystic lesion occupying much of the right lower lobe (Fig. 4). A considerable amount of calcareous material was seen within the cystic lesion and also in the supradiaphragmatic region and along the thoracic wall below and lateral to the cyst. No bronchovascular markings were seen in the latter area.

Right thoracotomy disclosed a tumor replacing most of the right lower lobe. Resection of the lobe was accomplished without difficulty and the patient recovered uneventfully.

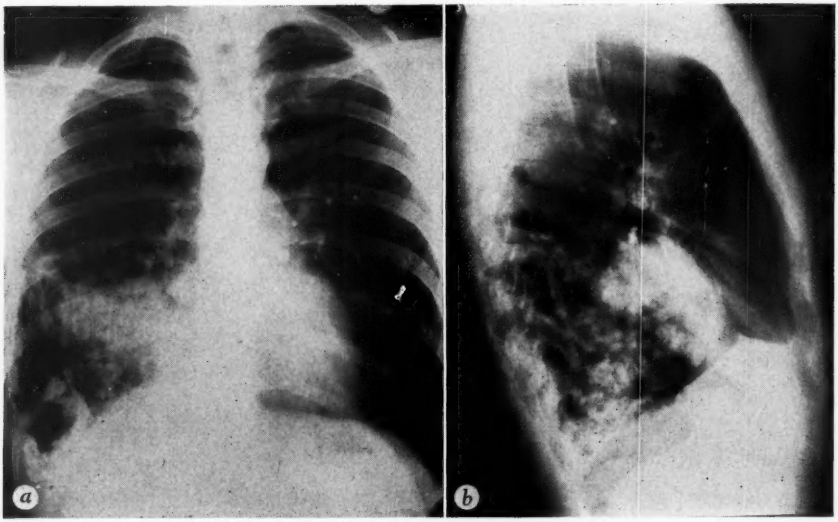

Fig. 4, *a* and *b*.—Case 2. Posteroanterior and right lateral roentgenographic views of hamartoma.

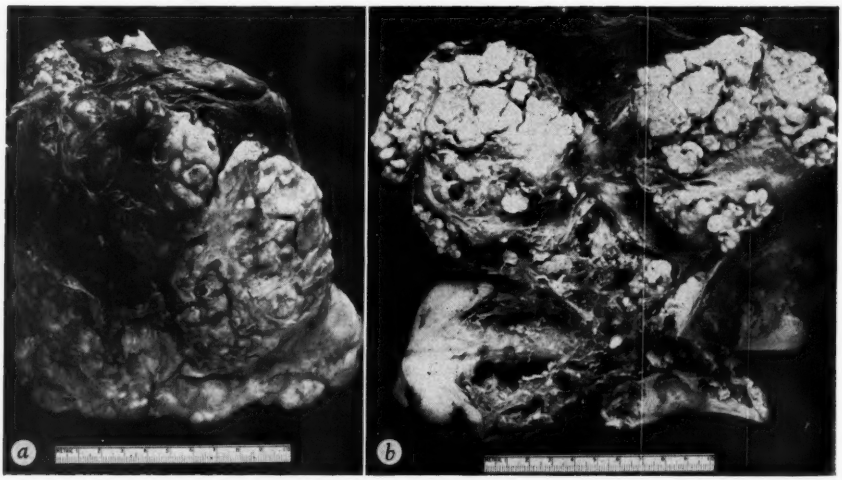

Fig. 5.—Case 2. *a*, Gross specimen removed at right lower lobectomy. *b*, Cross section illustrating multiloculated air-containing cysts.

The surgical specimen measured 16 by 14 by 10 cm. and weighed 800 grams (Fig. 5). It was bluish white and its surface was bosselated except for the undersurface, which was smooth and had the conformation of the diaphragm. The upper margin was covered by a layer of residual pulmonary tissue which stripped readily from the tumor. Many parts were of bony consistency but others were readily cut and had the appearance of hyaline cartilage. Small fluid-containing clefts were numerous. There were large multiloculated air-containing cysts in its center. Continuity with the normal bronchial system could not be demonstrated.

Microscopically, large islands and connecting strands of hyaline cartilage encircled by fibrous connective tissue predominated in the field (Fig. 6). Between these areas were cystic spaces of variable size lined with cuboid, columnar, pseudostratified columnar and occasionally mucus-producing epithelium. The cartilage was calcified in many areas. Mature fat and bone made up a lesser part of the lesion, and myxomatous connective tissue, smooth muscle, and mucosal glands were also identified. The large air-containing cysts were lined with columnar epithelium. Some areas strikingly resembled normal bronchi (Fig. 6, *b*). No alveolar structure was found within the confines of the tumor.

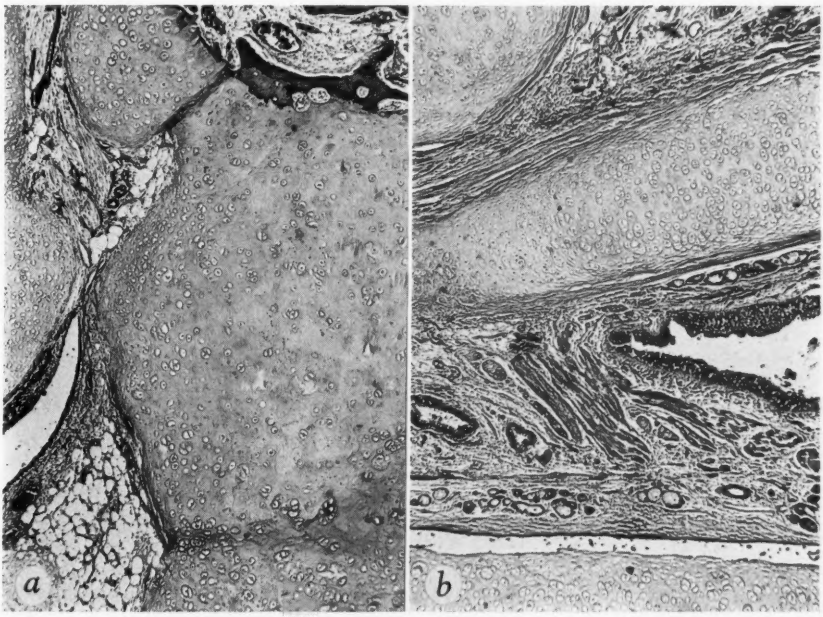

Fig. 6.—Case 2. *a*, Section showing typical pattern of the bulk of the lesion. (Hematoxylin and eosin; ×30.) *b*, Section showing a closer resemblance to normal bronchi, with a high degree of differentiation of tumor tissue. (Hematoxylin and eosin; ×40.)

In reviewing the literature, we found that a pulmonary hamartoma greater than 5 cm. in diameter is a medical rarity. Lesser, Linser, and Reboul,[7-9] each reported a case of benign hamartoma that measured in excess of 10 cm. in diameter. Schumann[10] described a hamartoma the "size of a man's head"; this tumor weighed 12 pounds (5.4 kilograms). The lesion reported by Busse[11] was huge, filling the entire hemithorax and exerting enough pressure to bulge the intercostal spaces. Careful analysis of his paper leaves considerable doubt as to the true nature of the tumor.

The premature infant reported by Jones[12] is of particular interest. Although the lesion measured only 3.5 cm. in diameter, it replaced the major portion of the right upper lobe. Cartilage and bronchial epithelium were present in small nests within islands of fibrous connective tissue separated by normal lung. Liebow[13] has referred to this lesion as a "diffuse hamartoma." We have seen no counterpart of it in adults and it seems reasonable to assume that, with growth of the lesion, the included pulmonary tissue could either be rendered functionless and made cystic or be overgrown to such an extent that it would be unrecognizable. If the growth differential is primarily in favor of the cartilage and epithelium in these malformations, the end result in Jones' case would have been identical to that in the 2 cases we have presented. Unfortunately, we have been unable to find another reported case of true hamartoma in an infant and, until these are available, the early development of hamartoma will remain in doubt.

The cystic nature of hamartomas deserves comment. Microscopic epithelium-lined clefts are characteristic of the lesions. The presence of small, grossly visible cysts is not unusual. McGlumphy[14] reported a hamartoma, 2 cm. in diameter, that contained a cyst 9 mm. in diameter. We did not find a case of benign pulmonary hamartoma with a large cyst or cysts in our review of the literature. A cystic component in these lesions may be suggested occasionally on the roentgenograms, but these "rarefied areas" usually prove to be fat tissue in the specimen. In both of the cases reported here, the masses contained large cysts and presented as cystic lesions clinically, one on the basis of roentgen findings and the other on the basis of a history of recurrent aspirations that were productive of large quantities of fluid.

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THE AMERICAN ASSOCIATION FOR THORACIC SURGERY

THIRTY-SIXTH ANNUAL MEETING
MAY 7, 8, AND 9, 1956
HOTEL FONTAINEBLEAU, MIAMI BEACH, FLA.

Program

MEMORANDA FOR GUIDANCE OF ESSAYISTS AND DISCUSSANTS

1. In accordance with the By-Laws of the Association the papers which are read at the Meeting shall be given to the Secretary *immediately after presentation.* If these papers are not submitted promptly their publication may well be delayed.
2. It is customary to group all of the discussions together on a series of papers. Any delay in the return of corrected discussions to the Editor means that publication of all papers on the subject will be held up. This is manifestly unfair to those who are conscientious in the prompt submission of their remarks for publication. Undue tardiness may mean that the discussion will not be published.
3. *Regular Program.*—Essayists (except in symposia) will be limited to 15 minutes for presentation.
4. *Thoracic Surgical Forum.*—Essayists will be limited to 10 minutes for their presentations. Papers on the Thoracic Surgical Forum as submitted for publication will be limited to 3,000 words. At the discretion of the President and within the limits of the allotted time, one or more discussions per paper will be allowed following all formal presentations.
5. *Discussants.*—Discussants will be limited to 3 minutes. Only members of the Association and Invited Speakers will have the privilege of discussing papers.

Monday Morning, May 7, 1956

8:30 A.M. Business Meeting.

9:00 A.M. Scientific Session: REGULAR PROGRAM.

1. **Congenital Diaphragmatic Hernia and Hypoplastic Lung: A Clinical Evaluation.** Benson B. Roe, San Francisco, Calif.
2. **Critically Crushed Chests: A New Method of Treatment by Internal "Pneumatic" Stabilization With Continuous Mechanical Hyperventilation.** Edward E. Avery (by invitation), E. Trier Mörch (by invitation), and Donald W. Benson (by invitation), Chicago, Ill.
3. **Complete Traumatic Rupture of the Left Main Bronchus Successfully Repaired Eleven Years After Injury.** Daniel E. Mahaffey (by invitation), Oscar Creech, Jr., Hollis G. Boren (by invitation), and Michael E. DeBakey, Houston, Texas.
4. **Congenital Atresia of Esophagus.** George H. Humphreys, Bruce M. Hogg (by invitation), and Jose Ferrer (by invitation), New York, N. Y.
5. **Torek Esophagectomy: The Case Against Segmental Resection for Esophageal Cancer.** William L. Watson, John T. Goodner (by invitation), Theodore P. Miller (by invitation), and George T. Pack (by invitation), New York, N. Y.

6. **Cardiac Surgery With Hypothermia and Acetyl Choline Arrest.** Peter V. Moulder (by invitation), Richard Thompson (by invitation), Curtis Smith (by invitation), and William E. Adams, Chicago, Ill.

Monday Afternoon, May 7, 1956

2:00 P.M. Scientific Session: REGULAR PROGRAM.

Symposium on Anesthesia and Related Problems (papers 7-11 inclusive)

7. **Contrast of Two Systems of Anesthesia for Thoracic Surgery.** Henry Beecher, Boston, Mass.
8. **A Consideration of Proper Methods in Thoracic Anesthesia.** E. M. Papper, New York, N. Y.
9. **The Cardiorespiratory Dynamics of Controlled Respiration in the Open and Closed Chest.** Archer S. Gordon (by invitation) and Charles W. Fyre (by invitation), Chicago, Ill.
10. **The Significance of the Lung-Thorax Compliance in Ventilation During Thoracic Surgery.** William E. Brownlee (by invitation) and Frank F. Allbritten, Jr., Kansas City, Kansas.
11. **Inefficient Carbon Dioxide Absorption Requiring Increased Pulmonary Ventilation During Operations With an Open Thoracotomy.** Thomas F. Nealon (by invitation), George J. Haupt (by invitation), Harold Chase (by invitation), and John H. Gibbon, Jr., Philadelphia, Pa.

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12. **Aortic Valvulotomy Under Direct Vision During Hypothermia.** F. John Lewis, Norman E. Shumway (by invitation), Suad A. Niazi (by invitation), and Robert B. Benjamin (by invitation), Minneapolis, Minn.
13. **The Indications for Lobectomy in the Treatment of Carcinoma of the Lung.** Joseph L. Robinson and John C. Jones, Los Angeles, Calif.
14. **Common Factors in Lung Cancer Survivors.** Richard H. Overholt and James A. Bougas (by invitation), Boston, Mass.

6:30 P.M.—8:30 P.M. COCKTAIL PARTY. Informal. Hotel Fontainebleau.

Tuesday Morning, May 8, 1956

8:30 A.M. Scientific Session: THORACIC SURGICAL FORUM.

15. **Introducing a Simple Surgical Method for the Correction of Mitral Regurgitation Using the Finger Ring Valve Elevator.** Aran S. Johnson (by invitation). Sponsored by William M. Tuttle, Detroit, Mich.
16. **Further Experiences With the Method of Controlled Unilateral Pulmonary Artery Occlusion in the Study of Lung Function.** Paul Nemir, Jr., H. H. Stone (by invitation), H. R. Hawthorne (by invitation), and T. N. Mackrell (by invitation), Philadelphia, Pa.
17. **Oxygen Availability to the Brain During Inflow Occlusion of the Heart in Normothermia and Hypothermia.** Samuel Kaplan (by invitation), Edward C. Matthews (by invitation), Louis Schwab (by invitation), and Leland C. Clark (by invitation). Sponsored by James A. Helmsworth, Cincinnati, Ohio.
18. **The Effect of Somatotrophin on Ventricular Fibrillation of Arterioclusive and Hypothermic Origin.** David H. Watkins (by invitation), S. Rothman (by invitation), Arthur E. Prevedel (by invitation), and Gordon A. Munro (by invitation). Sponsored by William B. Condon, Denver, Colo.
19. **Physiologic Responses in Man to Total Body Perfusion for Open Intracardiac Surgery.** Richard A. DeWall (by invitation) and Raymond C. Read (by invitation), Minneapolis, Minn.

20. **A Method for Controlled Cardiac Arrest as an Adjunct to Open Heart Surgery.** W. Glenn Young (by invitation), Will C. Sealy, Ivan W. Brown, Jr. (by invitation), Wilmer C. Hewitt, Jr. (by invitation), Henry A. Callaway, Jr. (by invitation), Doris H. Merritt (by invitation), and Jerome S. Harris (by invitation), Durham, N. C.
21. **The Neutralization of Heparin by Protamine in Extracorporeal Circulation.** Mr. Raymond Hurt (Travelling Fellow of the Association) (by invitation), London, England.
22. **Experience With a Disposable, Artificial Lung.** D. B. Effler, W. J. Kolff (by invitation), L. K. Groves (by invitation), F. M. Sones, Jr. (by invitation), and G. Peereboom (by invitation), Cleveland, Ohio.
23. **An Artificial Lung Dependent Upon Diffusion of Oxygen and Carbon Dioxide Through Plastic Membranes.** George H. A. Clowes, Jr., Amos L. Hopkins (by invitation), and William E. Neville (by invitation), Cleveland, Ohio.
24. **Extracorporeal Circulation for Open Heart Surgery.** Russell M. Nelson (by invitation), Hans H. Hecht (by invitation), Richard W. Hardy (by invitation), and Joe Burge (by invitation), Salt Lake City, Utah.
25. **A Mechanical Pump-Oxygenator for Direct Vision Repair of Atrial Septal Defects.** Bernard S. Levowitz (by invitation), Melvin M. Newman, Jackson H. Stuckey (by invitation), Marie C. Kernan (by invitation), Harry N. Iticovici (by invitation), and Clarence Dennis, Brooklyn, N. Y.
26. **Elimination and Transport of Mucus in the Lung: An Experimental Study in the Dog.** Edward G. Huppler (by invitation), O. Theron Clagett, and John H. Grindlay (by invitation), Rochester, Minn.
27. **Surgical Treatment of Ventricular Septal Defects: A Method Utilizing Selective Coronary Perfusion and Hypothermia.** John B. Grow, Charles V. Demong (by invitation), and Charles R. Hawes (by invitation), Denver, Colo.
28. **A Report on Heterologous Vascular Shunts (Bovine Brachiocephalic) in Experimental Aortic Arch Resection.** Ralph D. Alley, William H. Sewell (by invitation), Allan Stranahan, Harvey W. Kausel (by invitation), Thomas S. Reeve (by invitation), and Alan S. Peck (by invitation), Albany N. Y.
29. **Arterial Homograft Substitution and Bypass in Superior Vena Caval Obstruction.** John F. Higginson, Portland, Ore.
30. **Experiments With Substitute Esophagus.** Laurence Rubenstein (by invitation), Chicago, Ill.

Tuesday Afternoon, May 8, 1956

2:00 P.M. Executive Session. (Limited to Active and Senior Members.)

3:00 P.M. Scientific Session: REGULAR PROGRAM.

Address of the President, Richard H. Meade, Grand Rapids, Mich.

Some of the Forgotten Men in the Field of Thoracic Surgery.

Address of Honored Guest, Mr. Philip Allison, Ch.M., F.R.C.S., Nuffield Professor of Surgery, Oxford, England.

Observations on a Conservative Approach to Nonmalignant Lesions at the Cardia.

31. **Reconstruction of the Trachea.** John M. Keshishian (by invitation), Brian Blades, and Edward J. Beattie, Jr., Washington, D. C.
32. **The Solitary Pulmonary Nodule—A Ten-Year Survey.** Edgar W. Davis, J. Winthrop Peabody, Jr. (by invitation), and Sol Katz (by invitation), Washington, D. C.

7:00 P.M. COCKTAILS, BANQUET AND DANCING, Hotel Fontainebleau.
Attendance limited to members of the association and their ladies, invited speakers and their ladies.
Dinner dress preferred.

Wednesday Morning, May 9, 1956

9:00 A.M. Scientific Session: REGULAR PROGRAM.

33. **Pulmonary Resection for Tuberculosis: A Five- to Ten-Year Follow-up Study.** James D. Murphy and James M. Davis (by invitation), Oteen, N. C.
34. **The Role of the Chronic Occult Postresection Bronchial Fistula in the Reactivation of Tuberculosis: Pathogenesis and Treatment.** John W. Bell (by invitation) and E. M. Medlar (by invitation), Sunmount, N. Y.
35. **Extraperiosteal Plombage Thoracoplasty. Operative Technique and Results With 161 Cases With Unilateral Surgical Problems.** Norman J. Wilson, Orlando Armada (by invitation), William V. Vindzberg (by invitation), and William B. O'Brien (by invitation), Boston, Mass.
36. **Bronchographic Studies as a Guide in the Surgical Treatment of Pulmonary Tuberculosis.** F. J. Phillips, Anthony Lalli (by invitation), and Walther Buhler (by invitation), Bartlett, Alaska.
37. **A Clinical Evaluation of Decortication.** David H. Waterman, Sheldon E. Domm, and William K. Rogers (by invitation), Knoxville, Tenn.

Wednesday Afternoon, May 9, 1956

2:00 P.M. Scientific Session: REGULAR PROGRAM.

38. **Observations on the Surgical Pathology of Congenital Intracardiac Defects Made During Direct Vision Repair.** Herbert E. Warden (by invitation), Richard L. Varco, and C. Walton Lillehei, Minneapolis, Minn.
39. **Technique for Repair of Ventricular Septal Defects Utilizing Extracorporeal Circulation.** John W. Kirklin, H. G. Harshbarger (by invitation), D. E. Donald (by invitation), and J. E. Edwards (by invitation), Rochester, Minn.
40. **The Use of the Heart-Lung Apparatus in Human Cardiac Surgery.** F. D. Dodrill, Norman Marshall (by invitation), Jan Nyboer (by invitation), Elsie Noe (by invitation), C. H. Hughes (by invitation), and A. B. Stearns (by invitation), Detroit, Mich.
41. **The Treatment of Mitral Insufficiency by the "Purse-String" Technique.** Robert P. Glover, Julio C. Davila (by invitation), and O. Henry Janton (by invitation), Philadelphia, Pa.
42. **Mitral Insufficiency—Treatment by Polar Cross-Plication of the Annulus Fibrosus.** Henry T. Nichols (by invitation), Philadelphia, Pa.
43. **Patent Ductus Arteriosus in Infancy.** S. Richard Bauersfeld (by invitation), Paul C. Adkins (by invitation) and Edward M. Kent, Pittsburgh, Pa.